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THE PRINCIPLES AND PRACTICE OF MEDICINE

DESIGNED FOR THE USE OF
PRACTITIONERS AND STUDENTS OF MEDICINE

BY

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See note facing contents, 69

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TO
THE MEMORY OF MY TEACHERS:

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NOTE.

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JOHNS HOPKINS HOSPITAL,
BALTIMORE, *January 1, 1892.*

"Experience is fallacious and judgment difficult."
HIPPOCRATES: *Aphorisms, I.*

"And I said of medicine, that this is an art which considers the constitution of the patient, and has principles of action and reasons in each case."
PLATO: *Gorgias.*

Note: It is my impression that this word is misspelled in Plassue

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* The red shows the two-hourly, the black the morning and evening temperature.

A TEXT-BOOK ON THE PRACTICE OF MEDICINE.

SECTION I.

SPECIFIC INFECTIOUS DISEASES.

I. TYPHOID FEVER.

Definition.—An infectious disease, characterized anatomically by hyperplasia and ulceration of the lymph-follicles of the intestines, swelling of the mesenteric glands and spleen, and parenchymatous changes in the other organs. The bacillus of Eberth is constantly present in the lesions. Clinically the disease is marked by fever, a rose-colored eruption, diarrhœa, abdominal tenderness, tympanites, and enlargement of the spleen; but these symptoms are extremely inconstant, and even the fever varies in its characters.

Historical Note.—The dates 1813 and 1850 include the modern discussion of the subject. Prior to the former year many observers had noted clinical differences in the continued fevers. Huxham in particular, in his remarkable essay, had recognized varieties. In 1813 Pierre Bretonneau, of Tours, distinguished "dothiénentérite" as a separate disease; and Petit and Serres described entero-mesenteric fever. Trousseau and Velpeau, students of Bretonneau, were, in 1820, instrumental in making his views known to Andral and others in Paris. In 1829 Louis' great work appeared, in which the name "typhoid" was given to the fever. At this period typhoid fever alone prevailed in Paris, and it was universally believed to be identical with the continued fever of Great Britain, where in reality typhoid and typhus coexisted, and the intestinal lesion was regarded as an accidental occurrence in the course of ordinary typhus. Louis' students returning to their homes in different countries had opportunities of studying the prevalent fevers in the thorough and systematic manner of their master. Among these were certain young American physicians, to one of whom, Gerhard, of Philadelphia, is due the great honor of having first clearly laid down the differences between the two diseases. His papers in the American Journal of the Medical Sciences are undoubtedly the first in any language which give a full and

satisfactory account of the clinical and anatomical distinctions we now recognize. No student should fail to read these articles, among the most classical in American medical literature.

Louis' influence was early felt in Boston, to which, in 1833, James Jackson, Jr., had returned from Paris. In this year he demonstrated, in his father's wards at the Massachusetts General Hospital, the identity of the typhus of this country with the typhoid of Louis. He had already, in 1830, noticed the intestinal lesions in the common fever of New England. Though cut off at the very outset of his career, we may reasonably attribute to his inspiration the two elaborate memoirs on typhoid fever which, in 1838 and 1839, were issued from the Massachusetts General Hospital, by James Jackson, Sr., and Enoch Hale. These, with Gerhard's articles, contributed to make typhoid fever, as distinguished from typhus, widely recognized in the profession here long before the distinctions were recognized generally in Europe. Thus, the diseases were described under different headings in the first edition of Bartlett's admirable work on Fevers published in 1842.

The recognition in Paris of a fever distinct from typhoid, without intestinal lesions, was due largely to the influence of the able papers of George C. Shattuck, of Boston, and Alfred Stillé, of Philadelphia, which were read before the Société médicale d'Observation in 1838. At Louis' request, Shattuck went to the London Fever Hospital to study the disease in England, where he saw the two distinct affections, and brought back a report which was very convincing to the members of the society.

Stillé had the advantage of going to Paris knowing thoroughly the clinical features of typhus fever, for he had been Gerhard's house-physician at the Philadelphia Hospital, where he had studied during the epidemic of 1836. At La Pitié, with Louis, he saw quite a different affection, while in London, Dublin, and Naples he recognized typhus as he had seen it in Philadelphia. The results of his observation were given in an exhaustive paper which presented in tabular form the contrasts and distinctions, clinical and anatomical, which we now recognize.

In Great Britain the non-identity of typhus and typhoid was clearly established at Glasgow, where from 1836 to 1838 A. P. Stewart studied the continued fevers, and in 1840 published the results of his observations. In the decade which followed many important works were issued and more correct views gradually prevailed; but it was not until the publication of Jenner's observations between 1849 and 1851 that the question was finally settled in England.

Etiology.—Typhoid fever prevails especially in temperate climates, in which it constitutes the most common continued fever. Widely distributed throughout all parts of the United States and Canada, it probably presents everywhere the same essential character.

It prevails most in the autumn months. Of 1,889 cases admitted to the Montreal General Hospital in twenty years, more than fifty per cent

were in the months of August, September, and October. Of 1,381 cases treated during twelve years at the Toronto General Hospital, 761 occurred in these months (Graham). It has been well called the autumnal fever.

It has been observed to prevail most in hot and dry seasons. According to Pettenkofer, epidemics are most common when the ground-water is low, under which circumstances the springs and water-sources drain more thoroughly contaminated foci and are more likely to be highly charged with poison. It may be also, as Baumgarten suggests, that in dry seasons the poison is more disseminated by the dust.

Males and females are about equally liable to the disease, but males with typhoid are much more frequently admitted into hospitals.

Typhoid fever is a disease of youth and early adult life. The greatest susceptibility is between the ages of fifteen and twenty-five. Of 660 of the Montreal cases there were under fifteen years of age, 51; between fifteen and twenty-five years, 308; between twenty-five and thirty-five years, 153; between thirty-five and forty-five years, 43; between forty-five and fifty-five years, 6; and over fifty-five years, 9. Cases are rare over sixty. It is not very infrequent in childhood, but infants are rarely attacked. Murchison has seen a case at the sixth month. It is stated that the disease may be congenital in cases in which the mother has had the disease late in pregnancy.

As in other fevers, not all exposed to the infection take the disease, and there are grades of susceptibility. Some families seem more disposed to infection than others.

The Specific Germ.—The researches of Eberth, Koch, Gaffky, and others have shown that there is a special micro-organism *constantly* associated with typhoid fever. It is a rather short, thick, motile bacillus, with rounded ends, in one of which, sometimes in both (particularly in cultures), there can be seen a glistening round body, believed to be a spore; but these polar structures are probably only areas of dense protoplasm. It grows readily on various nutritive media, and on potato in a characteristic manner, as the growth is invisible. This feature is not peculiar however to the typhoid bacillus. It is difficult to differentiate from the *bacterium coli commune*, except by certain chemical tests. This organism fulfils two of the requirements of Koch's law—it is constantly present, and it grows outside the body in a specific manner. The third requirement, the production of the disease experimentally by the cultures, has not yet been met. Probably the animals used for experimentation are not susceptible to typhoid fever. The bacilli inoculated in large quantities into the blood of rabbits are pathogenic, and in some instances ulcerative and necrotic lesions in the intestine may be produced. But similar intestinal lesions may be caused by other bacteria, including the *bacterium coli commune.*

The bacilli produce various poisons, of which Brieger has described a ptomaine—typhotoxin, and Brieger and Fränkel a toxalbumin; but our

information on these substances is still very defective. Cultures are killed at a temperature of 60° C. It is not probable that the typhoid bacillus produces spores, but it resists drying for days. Bouillon cultures are destroyed by carbolic acid, 1 to 200, and by corrosive sublimate, 1 to 2,500.

In recent cases of typhoid fever the bacillus is found in the lymphoid tissues of the intestines, in the mesenteric glands, in the spleen, and in the liver. It occurs also in irregular clumps in the contents of the intestines and in the stools. The bacillus is said to have been found rarely in the blood, in the rose-colored spots (?), and in the urine.

Outside the body the bacilli retain their vitality for weeks in water. Whether an increase can occur is not yet finally settled. Bolton denies it, but the general opinion seems to be that such increase may take place to some extent. They disappear from ordinary water in competition with saprophytes in a few days. In milk they undergo rapid development without changing the appearance of the milk. They may increase in the soil and retain their vitality for months. They are not killed by freezing, but, as Prudden has shown, may live in ice for months. In many epidemics the bacilli have been detected in the infected water. The detection however of the typhoid bacillus in drinking-water is by no means easy, and the question in individual cases must be settled by experts who have had special experience with this germ. Both Prudden and Ernst have found it in water-filters.

Modes of Conveyance.—(*a*) *Contagion.*—Typhoid fever is certainly not a very contagious disease, but the possibility of direct transmission must be acknowledged. The poison is not given off from the skin or in the breath, but in the fæces. Practically only those persons are liable to contract the disease in this way who have to do with the stools or with the body-linen of patients. I have known several instances in which nurses appear to have been infected under these conditions.

(*b*) *Infection of water* is unquestionably the most common mode of conveyance. Many epidemics have been shown to originate in the contamination of a well or a spring. A very striking one occurred at Plymouth, Pa., in 1885, which was investigated by Shakespeare. The town, with a population of eight thousand, was in part supplied with drinking-water from a reservoir fed by a mountain stream. During January, February, and March, in a cottage by the side of and at a distance of from sixty to eighty feet from this stream, a man was ill with typhoid fever. The attendants were in the habit at night of throwing out the evacuations on the ground toward the stream. During these months the ground was frozen and covered with snow. In the latter part of March and early in April there was considerable rainfall and a thaw, in which a large part of the three months' accumulation of discharges was washed into the brook, not sixty feet distant. At the very time of this thaw the patient had numerous and copious discharges. About the 10th of April cases of typhoid fever broke out in the town, appearing for a time at the rate of fifty a

day. In all about twelve hundred people were affected. An immense majority of all the cases were in the part of the town which received water from the infected reservoir.

Milk also may be the source of infection. One of the most thoroughly studied epidemics due to this cause was that investigated by Ballard in Islington. The milk may be contaminated by infected water used in cleansing the cans. In fresh milk it has been shown that the germs grow rapidly.

Filth, bad sewers, or cesspools can not in themselves cause typhoid fever, but they furnish the conditions suitable for the preservation of the bacillus and possibly for its propagation.

(*c*) *Contamination of the Soil.*—Pettenkofer holds that the poison is not eliminated in a condition capable of communicating the disease directly, but that it must first undergo changes in the soil, which changes are favored by the ground-water.

It does not seem probable that typhoid fever is communicated by the air alone, as by the medium of sewer-gas.

Once in the intestinal canal the typhoid germs probably do not like the cholera bacilli increase in the secretions, but penetrate the epithelial lining and reach the lymphoid tissue, upon which they exert their specific action, causing a cell proliferation greatly in excess of the physiological process. The necrosis may be regarded as the result of the maximum intensity of the action of the bacilli—an action not confined to the lymphatic apparatus of the intestinal wall, but also met with in a typical manner in the enlarged mesenteric glands and in the liver and spleen.

It has not yet been definitely determined whether the constitutional disturbances in typhoid fever depend upon the toxalbumins produced in the growth of the bacilli, though this is in the highest degree probable.

Morbid Anatomy.—The statistical details under this heading are based upon sixty-four autopsies, a majority of which were performed at the Montreal General Hospital, and upon the records of two thousand post-mortems at the Munich Pathological Institute.*

Intestines.—A catarrhal condition exists throughout the small and large bowel, and to this is due, in all probability, the diarrhœa with the thin pea-soup-like stools. Associated with this catarrh there is during life some epithelial desquamation.

Specific changes occur in the lymphoid elements of the bowel, chiefly at the lower end of the ileum. The alterations which occur are most conveniently described in four stages:

1. *Hyperplasia*, which involves the glands of Peyer in the jejunum and ileum, and to a variable extent those in the large intestine. The follicles are swollen, grayish-white in color, and the patches may project to a distance of from three to five mm. In exceptional cases they may be still more

* Münchener medicinische Wochenschrift, Nos. 3 and 4, 1891.

prominent. The solitary glands, which range in size from a pin's head to a large pea, are usually deeply imbedded in the submucosa, but project to a variable extent. Occasionally they are very prominent and may be almost pedunculated. Microscopical examination shows at the outset a condition of hyperæmia of the follicles. Later there is a great increase and accumulation of cells of the lymph-tissue which may even infiltrate the adjacent mucosa and the muscularis; and the blood-vessels are more or less compressed, which gives the whitish anæmic appearance to the follicles. The cells have all the characters of ordinary lymph-corpuscles. Some of them however are larger, epithelioid, and contain several nuclei. Occasionally cells containing red blood-corpuscles are seen. This so-called medullary infiltration, which is always more intense toward the lower end of the ileum, reaches its height from the eighth to the tenth day and then undergoes one of two changes, *resolution* or *necrosis*. Death very rarely takes place at this stage. I have seen but one instance in my series—a girl, aged twenty-four, who died at the end of the first week with severe nervous symptoms and in whose ileum the lymph-follicles were greatly swollen, pitted and cribriform, but without necrosis. Resolution is accomplished by a fatty and granular change in the cells, which are destroyed and absorbed. A curious condition of the patches is produced at this stage, in which they have a reticulated appearance, the *plaques à surface réticulée*. The swollen follicles in the patch undergo resolution and shrink more rapidly than the surrounding framework, or what is more probable the follicles alone owing to the intense hyperplasia become necrotic and disintegrate leaving the little pits. In this process superficial hæmorrhages may result and small ulcers may originate by the fusion of these superficial losses of substance.

There is nothing distinctive in the hyperplasia of the lymph-follicles in typhoid fever. Apart from this disease we rarely see in adults a marked affection of these glands with fever. In children however it is not uncommon when death has occurred from intestinal affections. It is also met with in measles, diphtheria, and scarlet fever.

2. *Necrosis and Sloughing.*—When the hyperplasia of the lymph-follicles reaches a certain grade resolution is no longer possible. The blood-vessels become choked, there is a condition of anæmic necrosis, and sloughs form which must be separated and thrown off. The necrosis is probably due in great part to the direct action of the bacilli. The process may be superficial, affecting only the upper part of the mucous coat, or it may extend to and involve the submucosa. It is always more intense toward the ileo-cæcal valve, and in very severe cases the greater part of the mucosa of the last foot of the ileum may be converted into a brownish-black eschar. The necrosis in the solitary glands forms a yellowish cap which often involves only the most prominent point of a follicle. The extent to which the necrosis reaches is very variable. It may pass deep into the muscular coat reaching to or even perforating the peritonæum.

3. *Ulceration.*—The separation of the necrotic tissue—the sloughing—is gradually effected from the edges inward, and results in the formation of an ulcer, the size and extent of which are directly proportionate to the amount of necrosis. If this be superficial, the entire thickness of the mucosa may not be involved and the loss of substance may be small and shallow. More commonly the slough in separating exposes the submucosa and muscularis, particularly the latter, which forms the floor of a majority of all typhoid ulcers. It is not common for an entire Peyer's patch to slough away, and a perfectly ovoid ulcer opposite to the mesentery is rarely seen. Irregularly oval and rounded forms are most common. A large patch may present three or four ulcers divided by septa of mucous membrane. The terminal six or eight inches of the mucous membrane of the ileum may form a large ulcer, in which are here and there islands of mucosa. The edges of the ulcer are usually swollen, soft, sometimes congested, and often undermined. At a late period the ulcers near the valve may have very irregular sinuous borders. The base of a typhoid ulcer is smooth and clean, usually formed of the submucosa or of the muscularis.

There may be large ulcers near the valve and swollen hyperæmic patches of Peyer in the upper part of the ileum.

4. *Healing.*—This begins with the development of a thin granulation tissue which covers the base and gives to it a soft, shining appearance. The mucosa gradually extends from the edge, and a new growth of epithelium is formed. The glandular elements are reformed; the healed ulcer is somewhat depressed and is usually pigmented. Occasionally an appearance is seen as if an ulcer had healed in one place and was extending in another. In death during relapse healing ulcers may be seen in some patches with fresh ulcers in others.

We may say, indeed, that healing begins with the separation of the sloughs, as, when resolution is impossible, the removal of the necrosed part is the first step in the process of repair. Practically, in fatal cases, we seldom meet with evidences of cicatrization, as the majority of deaths occur before this stage is reached.

Large Intestine.—The cæcum and colon are affected in about one third of the cases (in nineteen of the sixty-four). Sometimes the solitary glands are greatly enlarged. The ulcers are usually larger in the cæcum than in the colon. Perforation of the cæcum is rare. The appendix may be involved. In my cases there was ulceration in two and perforation in one case. I dissected a case in Montreal in which the patient died three months after an attack of typhoid fever, and a localized abscess was found, due to perforation of the appendix. Death resulted from pylephlebitis.

Perforation of the Bowel.—In one hundred and fourteen cases of the two thousand Munich autopsies (5·7 per cent) and in fourteen instances in my series, the intestine was perforated and death caused by peritonitis. The perforation may occur in ulcers from which the sloughs have already

separated, or it may be directly due to the extension of a necrosis through all the coats. In only a few cases is the perforation at the bottom of a clean thin-walled ulcer. In one instance the perforation occurred two weeks after the temperature had become normal. The sloughs were, as a rule, adherent about the site of perforation. A majority of the cases were in small deep ulcers. There may be two or even three perforations. The orifice is usually within the last foot of the ileum. In only one of my cases was it distant eighteen inches. Peritonitis was present in every instance.

Hæmorrhage from the bowels occurred in ninety-nine of the Munich cases, and in nine of my series. The bleeding seems to result directly from the separation of the sloughs. I was not able in any instance to find the bleeding vessel. In one case only a single patch had sloughed, and a firm clot was adherent to it. The bleeding may also come from the soft swollen edges of the patch.

The *mesenteric glands* at first show intense hyperæmia and subsequently become greatly swollen. Spots of necrosis are common. In several of my cases suppuration had occurred. The bunch of glands in the mesentery, at the lower end of the ileum, is especially involved. The retroperitoneal glands are also swollen.

The *spleen* is invariably enlarged in the early stages of the disease. In only one of my cases did it exceed (600 grammes) 20 ounces in weight. The tissue is soft, even diffluent. Infarction is not infrequent. Rupture may occur spontaneously or as a result of injury. In the Munich autopsies there were five instances of rupture of the spleen, one of which resulted from a gangrenous abscess.

The *liver* shows signs of parenchymatous degeneration. Early in the disease it is hyperæmic, and in a majority of instances it is swollen, somewhat pale, on section turbid, and microscopically the cells are very granular and loaded with fat. Necrotic areas occur in many cases, as described by Handford. They have been studied recently by Reed in Welch's laboratory. No definite association could be determined between the groups of bacilli and the necrotic areas. In twelve of the Munich autopsies liver abscess was found, and in three, acute yellow atrophy. Diphtheritic inflammation of the gall-bladder is occasionally met with. This may lead to perforation and fatal peritonitis.

The *kidneys* show cloudy swelling, with granular degeneration of the cells of the convoluted tubules; less commonly an acute nephritis. A rare condition described by Rayer, Wagner, and others is the occurrence of numerous small areas infiltrated with round cells, which may have the appearance of lymphomata (Wagner), or may pass on to softening and suppuration, producing the so-called *miliary abscesses*. It is usually a late change. The bacilli have been found by some observers in these areas. The bacilli can be obtained by culture from the kidneys, and have been found in many instances in sections. They have also been found in

the urine in a few cases. Diphtheritic inflammation of the pelvis of the kidney may occur. It was present in three of my cases, in one of which the tips of the papulæ were also affected. Catarrh of the bladder is not uncommon. Diphtheritic inflammation of it may also occur. Orchitis is occasionally met with.

The anatomical changes in the *respiratory organs* are not very numerous. Ulceration of the larynx occurs in a certain number of cases; in the Munich series it was noted one hundred and seven times. It may come on at the same time as the ulceration in the ileum, but the bacilli have not yet, I believe, been found in the ulcers. They occur in the posterior wall, at the insertion of the cords, at the base of the epiglottis, and on the ary-epiglottidean folds. In the later periods catarrhal and diphtheritic ulcers may be present.

Œdema of the glottis was present in twenty of the Munich cases, in eight of which tracheotomy was performed. Diphtheritic laryngitis is not very uncommon. It occurred in a most extensive form in two of my cases. In one the membrane was chiefly in the pharynx, and extended only upon the epiglottis; in the other there was a uniform membrane which extended into the trachea and in the tubes of the second dimension. In eight cases in my series there was lobar pneumonia. Hypostatic congestion and the condition of the lung spoken of as splenization are very common. Gangrene of the lung occurred in forty cases in the Munich series; abscess of the lung in fourteen; hæmorrhagic infarction in one hundred and twenty-nine. Pleurisy is not a very common event. Fibrinous pleurisy occurred in about six per cent of the Munich cases, and empyema in nearly two per cent.

Changes in the Circulatory System.—Endocarditis is rare. It was not present in any of my cases, and existed in eleven only of the Munich autopsies, in which also there were fourteen cases of pericarditis. Myocarditis is not very infrequent. Dewevre,* in a series of forty-eight cases, found in sixteen granular or fatty degeneration, and in three a proliferating endarteritis in the small vessels. It is remarkable that even in cases of death from heart-failure, with intense fever, the cell-fibres may present little or no observable change. The *arteries* are not infrequently involved in typhoid fever. Barié distinguishes an acute obliterating arteritis and a partial arteritis, and states that they both occur most commonly in the arteries of the lower extremities. They are responsible, no doubt, for certain of the cases of blocking of the arterial trunks. This arteritis may affect the smaller vessels, particularly those of the heart. In the veins, thrombi are not infrequently found, particularly in the femoral veins, and more rarely in the cerebral sinuses.

Nervous System.—There are very few coarse changes met with. Meningitis is extremely rare. It was not present in any one of my autop-

* Archives générales de Médecine, 1887, 2.

sies, and occurred in only eleven of the two thousand Munich cases. The anatomical lesion upon which the aphasia—seen not infrequently in children—depends, is not known. Possibly, as Leyden states, it may be due to slight encephalitis. Parenchymatous changes have been met with in the peripheral nerves, and appear to be not very uncommon, even when there have been no symptoms of neuritis.

The voluntary muscles show, in certain instances, the peculiar changes described by Zenker which occur in all long-standing febrile affections and are not peculiar to typhoid fever. The muscle substance within the sarcolemma undergoes either a granular degeneration or a hyaline transformation. The abdominal muscles, the adductors of the thighs, and the pectorals are most commonly involved.

Symptoms.—In a disease so complex as typhoid fever it will be well first to give a general description and then to study more fully the sypmtoms, complications, and sequelæ according to the individual organs.

General Description.—The period of incubation lasts from a week to ten days, during which there are feelings of lassitude and inaptitude for work. The onset is rarely abrupt. There may be prodromal symptoms, either a rigor, which is rare, or chilly feelings, headache, nausea, loss of appetite, pains in the back and legs, and nose-bleeding. These symptoms increase in severity and the patient at last takes to his bed. From this event, in a majority of cases, the definite onset of the disease may be dated. During the *first week* there is, in some cases (but by no means in all, as has long been taught), a steady rise in the fever, the evening record rising a degree or a degree and a half higher each day, reaching 103° or 104°. The pulse is rapid, from 100 to 110, full in volume, but of low tension and often dicrotic; the tongue is coated and white; the abdomen is slightly distended and tender. Unless the fever is high there is no delirium, but the patient complains of headache, and there is mental confusion and wandering at night. The bowels may be constipated, or there may be two or three loose movements daily. Toward the end of the week the spleen becomes enlarged and the rash appears in the form of rose-colored spots, seen first on the skin of the abdomen. Cough and bronchitic symptoms are not uncommon at the outset.

In the *second week*, in cases of moderate severity, the symptoms become aggravated; the fever remains high and the morning remission is slight. The pulse is rapid and has lost its dicrotic character. There is no longer headache, but there is mental torpor and dulness. The face looks heavy; the lips are dry; the tongue, in severe cases, becomes dry also. The abdominal symptoms are more marked—diarrhœa, tympanites, and tenderness. Death may occur during this week, with pronounced nervous symptoms, or, toward the end of it, from hæmorrhage or perforation. In mild cases the fever declines, and by the fourteenth day may be normal.

In the *third week*, in cases of moderate severity, the pulse ranges from

110 to 130; the temperature now shows marked morning remissions, and there is a gradual decline in the fever. The loss of flesh is now more noticeable, and the weakness is pronounced. The diarrhœa and meteorism may persist. Unfavorable symptoms at this stage are the pulmonary complications, increasing feebleness of the heart, and pronounced delirium with muscular tremor. Special dangers are perforation and hæmorrhage.

With the *fourth week*, in a majority of instances, convalescence begins. The temperature gradually reaches the normal point, the diarrhœa stops, the tongue cleans, and the desire for food returns. In severe cases the fourth week may present an aggravated picture of the third; the patient grows weaker, the pulse is more rapid and feeble, the tongue dry, and the abdomen distended. He lies in a condition of profound stupor, with low muttering delirium and subsultus tendinum, and passes the fæces and urine involuntarily. Heart-failure and secondary complications are the chief dangers of this period.

In the *fifth and sixth weeks* protracted cases may still show irregular fever, and convalescence may not set in until after the fortieth day. In this period we meet with relapses in the milder forms or slight recrudescence of the fever. At this time, too, occur many of the complications and sequelæ.

Special Features and Symptoms.—*Mode of Onset.*—As a rule, the symptoms develop insidiously, and the patient is unable to fix definitely the time at which he began to feel ill. The following are the most important deviations from this common course:

(*a*) *Onset with Pronounced Nervous Manifestations.*—Headache, of a severe and intractable nature, is by no means an infrequent initial symptom. Again, a severe facial neuralgia may for a few days put the practitioner off his guard. In cases in which the patients have kept about and, as they say, fought the disease, the very first manifestations may be pronounced delirium. Such patients may even leave home and wander about for days. In rare cases the disease sets in with the most intense cerebro-spinal symptoms, simulating meningitis—severe headache, photophobia, retraction of the head, twitching of the muscles, and even convulsions. Occasionally drowsiness, stupor, and signs of basilar meningitis may exist for ten days or more before the characteristic symptoms develop; occasionally the onset is with mania.

(*b*) *With Pronounced Pulmonary Symptoms.*—The initial bronchial catarrh may be of great severity and disguise the other features of the disease. More striking still are those cases in which the disease sets in with a single chill, with pain in the side and all the characteristic features of lobar pneumonia.

(*c*) *With Intense Gastro-intestinal Symptoms.*—The vomiting may be incessant and uncontrollable. Occasionally there are cases with such intense vomiting and diarrhœa that a suspicion of poisoning may be aroused.

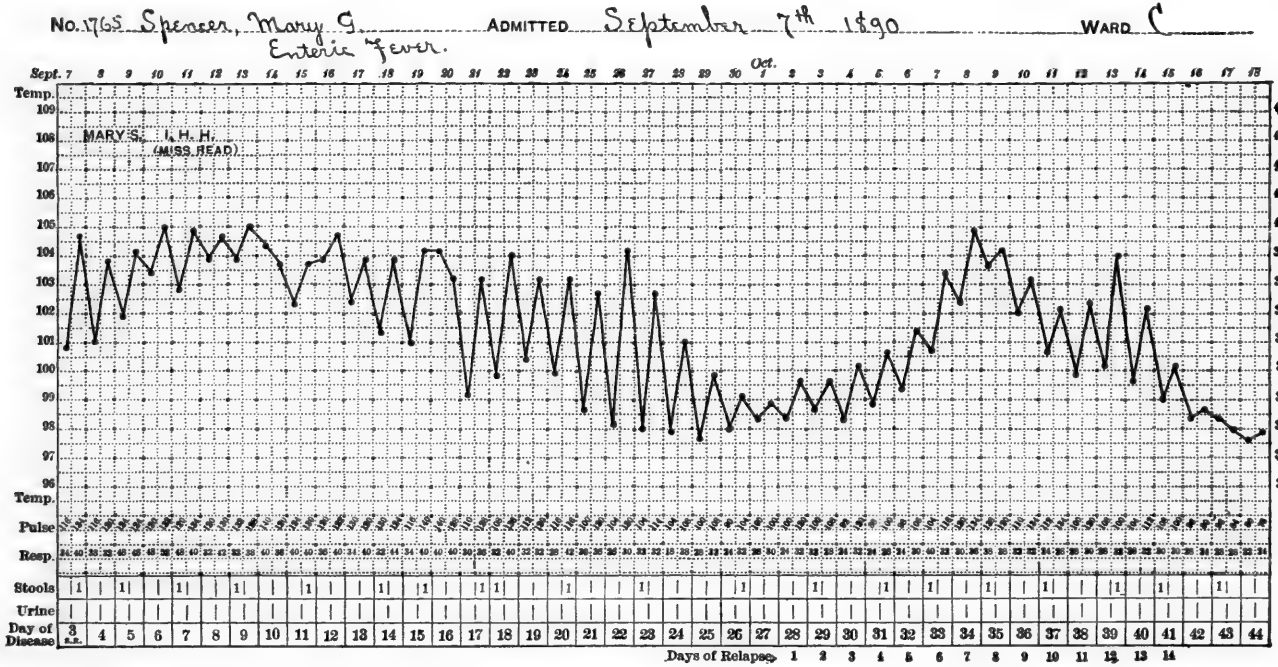

CHART I.—Typhoid Fever with relapse.

(*d*) *With Symptoms of an Acute Nephritis.*—Smoky or bloody urine, with much albumen and tube-casts.

(*e*) *Ambulatory Form.*—Deserving of especial mention are those cases of typhoid fever in which the patient keeps about and attempts to do work, or perhaps takes a long journey to his home. He may come under observation for the first time with a temperature of 104° or 105°, and the rash well out. Such cases seem always to run a more severe course than others, and in general hospitals they contribute largely to the total mortality. Finally, there are rare instances in which the first symptoms are perforation, or a profuse hæmorrhage from the bowels.

Facial Aspect.—Early in the disease the cheeks are flushed and the eyes bright. Toward the end of the first week the expression becomes more listless, and when the disease is well established the expression is dull and heavy.

Fever.—(*a*) *Regular Course.* (Chart I.)—In the stage of invasion the temperature may rise steadily during the first five or six days. The evening temperature is about a degree or a degree and a half higher than the morning remission, so that a temperature of 104° or 105° is not uncommon by the end of the first week. Having reached the fastigium or height, the fever then persists with slight morning remissions. The temperature curve follows the normal diurnal variations, the maximum occurring between four and eight o'clock in the evening and the minimum between four and eight in the morning. At the end of the second and throughout the third week the temperature becomes more distinctly remittent. The difference between the morning and evening may be three or four degrees, and the morning temperature may even be normal. It falls by gradual lysis, and the temperature is not considered normal until the evening record is at 98·2°.

(*b*) Variations in the normal temperature curve are common. We do not always see the gradual step-like ascent in the early stage; the cases do not often come under observation at this time. When the disease sets in with a chill, the temperature may rise at once to 103° or 104°. In many cases defervescence occurs at the end of the second week and the temperature may fall rapidly, reaching the normal within twelve or twenty hours. An inverse type of temperature, high in the morning and low in the evening, is occasionally seen but has no especial significance.

Sudden falls in the temperature may occur; thus, as shown in Chart IV, a drop of 10° may follow an intestinal hæmorrhage, and the fall may be very apparent even before the blood has appeared in the stools. Hyperpyrexia, temperature above 106°, is not very common in typhoid fever except just before death, when I have known the thermometer to register 109·5°. (Chart II.)

(*c*) *Post-Typhoid Elevations—Fever of Convalescence.*—During convalescence, after the temperature has been normal, perhaps for five or six days, the fever may rise suddenly to 102° or 103°, and, after per-

sisting for from one to three days or even longer, falls to normal. With this there is no constitutional disturbance, no furring of the tongue, no distention of the abdomen. These so-called recrudescences are by no means uncommon, and are of especial importance, as they cause great anxiety to the practitioner. They are attributed most frequently to errors in diet, constipation, emotions, and excitement of any sort, such as seeing friends.

There are cases in which the temperature declines almost to the normal at the end of the third week, the tongue cleans, and the patient enters

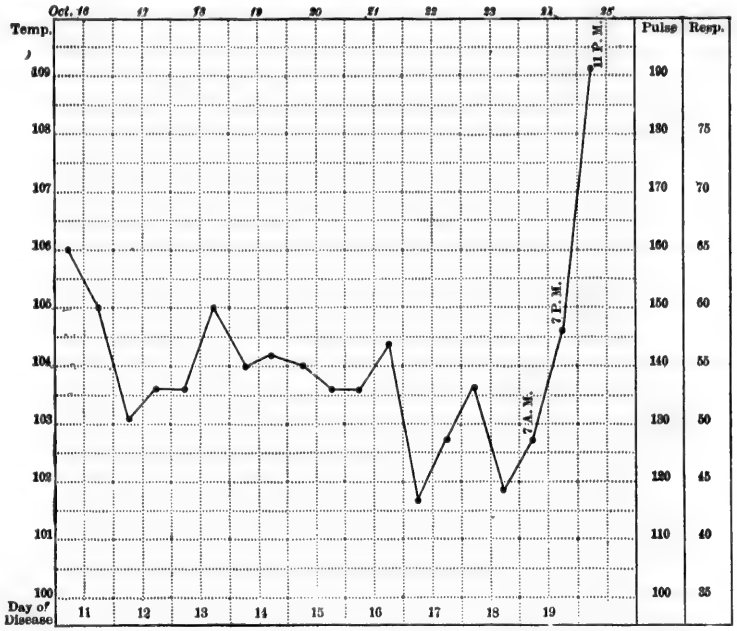

CHART II.—Hyperpyrexia—death.

apparently upon a satisfactory convalescence. The evening temperature, however, does not reach 98·5°, but constantly keeps about 99·5° or 100°, and occasionally rises to 100·5°. This, in the late stages of convalescence, I have seen due to the post-typhoid anæmia. Complications should be carefully looked for, particularly insidious pleurisy or bone lesions.

In certain of these cases the persistence of the fever seems to be really a nervous phenomenon, and there is nothing in the condition of the patient to cause uneasiness except the evening elevation of temperature. If the tongue is clean, the appetite good, and there are no intestinal symptoms, it may be disregarded. I have frequently found this condition best met by allowing the patient to get up and by stopping the use of the thermometer. This prolonged slight elevation of the fever after the dis-

appearance of all the symptoms is most common in children and in patients of marked nervous temperament.

(*d*) *The Fever of the Relapse.*—This is a repetition in many instances of the original fever, a gradual ascent and maintenance for a few days at a certain height and then a gradual decline. It is shorter than the original pyrexia, and rarely continues more than two or three weeks. (Chart I.)

(*e*) *Afebrile Typhoid.*—There are cases described in which the chief features of the disease have been present without the existence of fever. They are extremely rare in this country. No instance of the kind has come under my observation.

Skin.—The rash of typhoid fever is very characteristic. It consists of a number of rose-colored spots, which appear from the seventh to the tenth day, usually first upon the abdomen. The spots are flattened papules, slightly raised, of a rose-red color, disappearing on pressure, and ranging in diameter from two to four millimetres. They can be felt as distinct elevations on the skin. Sometimes each spot is capped by a small vesicle. The spots may be dark in color and occasionally become petechial. After persisting for two or three days they gradually disappear, leaving a brownish stain. They come out in successive crops, but rarely appear after the middle of the third week. They are present in the typical relapse. The rash is most abundant upon the abdomen and lower thoracic zone and often abounds upon the back. It is extremely variable in degree. There are cases in which it spreads to the extremities and often to the face. I can not say that in my experience these cases with the more abundant eruption have been of specially severe type. The rash is not always present. Murchison states that it is frequently absent in children.

A branny desquamation is not rare in cases in which the sudaminal vesicles have been abundant; occasionally the skin may peel in large flakes.

The following accidental rashes are met with in typhoid fever:

1. *Erythema.*—It is not very uncommon in the first week of typhoid fever to find the skin of a vivid red color, almost like a scarlatinal rash. This is particularly noticeable on the abdomen and chest, but the rash may spread to the extremities. It may possibly in some instances, but certainly not always, be due to quinine. I have seen it much more frequently in the past five years (during which time I have rarely ordered a dose of quinine in this disease) than I did in Montreal, where we used quinine largely as an antipyretic.

2. The *tache bleuâtre—Peliomata.*—These are pale-blue spots, subcuticular, from 4 to 10 mm. in diameter, of irregular outline and most abundant about the chest, abdomen, and thighs. They sometimes give a very striking appearance to the skin. It can be readily seen that the injection is in the deeper tissues and not superficial. This rash is quite without significance. Since my attention was called to its association with

body lice, I have met with no instance in which these were not present. Several French observers maintain that they are due to the irritating effects of the fluid secreted by pediculi.

3. Sudaminal and miliary eruptions are common in all cases in which there is profuse sweating.

4. Urticaria is occasionally met with, and lastly herpes, but this is uncommon in comparison with its frequency in malaria and pneumonia.

The *tache cérébrale*, a red line with white borders, can be produced by drawing the nail over the skin. It is a vaso-motor phenomenon which, as in other fevers, can be readily elicited, particularly in nervous subjects. Here may be mentioned certain other cutaneous phenomena also of vaso-motor nature: thus exposure of the abdomen may be sufficient to cause a pinkish injection, which may in places change to an ivory white, giving a curious mottled appearance to the skin. A similar appearance may be seen on the arms. The general tint may be white, with irregular patches or streaks of pink or dark red.

Sweats.—At the height of the fever the skin is usually dry. Profuse sweating is rare, but it is not very uncommon to see the abdomen or chest moist with perspiration, particularly in the reaction which follows the bath. Sweats in some instances constitute a striking feature of the disease. They may occasionally be associated with chilly sensations or actual chills. Jaccoud and others in France have especially described this *sudoral* form of typhoid fever. There may be recurring paroxysms of chill, fever, and sweats (even several in twenty-four hours), and the case may be mistaken for one of intermittent fever. The fever toward the end of the second week and during the third week may be intermittent. The characteristic rash is usually present, and if absent the negative condition of the blood is sufficient to exclude malaria. I have seen cases of this form in Montreal, where there could have been no suspicion of malarial infection.

Œdema of the skin occurs:

1. As the result of vascular obstruction, most commonly of a vein, as in thrombosis of the femoral vein.

2. In connection with nephritis.

3. In association with the anæmia and cachexia.

The hair is very apt to fall out after an attack of typhoid fever. Instances of permanent baldness are of extreme rarity. As in other diseases associated with fever the nutrition of the nails suffers, and during and after convalescence a transverse ridge is seen.

And, lastly, it is stated that a peculiar odor is exhaled from the skin in typhoid fever. Whether due to a cutaneous exhalation or not, there certainly is a very distinctive smell connected with many patients. I have repeatedly had my attention directed to it by nurses. Nathan Smith describes it as of a "semi-cadaverous, musty character."

Circulatory System.—The *blood* presents important changes. The

following statements are based on studies which W. S. Thayer has made in my ward. During the first two weeks there may be little or no change in the blood. Profuse sweats or copious diarrhœa may, as Hayem has shown, cause the corpuscles—as in the collapse stage of cholera—to rise above normal. In the third week a fall usually takes place in corpuscles and hæmoglobin and the number may sink rapidly even to 1,300,000 per c. mm., gradually rising to normal during convalescence. When the patient first gets up, there may be a slight fall in the number of the corpuscles.

The amount of hæmoglobin is always reduced, and usually in a greater relative proportion than the number of red corpuscles, and during recovery the normal color standard is reached at a later period. The number of colorless corpuscles varies little from the normal standard (6,000 ± per c. mm.). As a rule, perhaps the number is slightly subnormal (Pée). This fact is important, and may be at times of real diagnostic value in distinguishing typhoid fever from various septic fevers and acute inflammatory processes in which there is leucocytosis.

The accompanying blood-chart shows these changes well.

The post-typhoid anæmia may reach an extreme grade. In one of my cases the blood-corpuscles sank to 1,300,000 per cubic mm. and the hæmoglobin to about twenty per cent. These severe grades of anæmia are not common in my experience. In the Munich statistics there were fifty-four cases with general and extreme anæmia.

Of changes in the blood plasma very little is known.

The *pulse* in typhoid fever presents no special characters. It is increased in rapidity in proportion to the height of the fever. As a rule, in the first week it is above 100, full in volume and often dicrotic. There is no acute disease with which, in the early stage, a dicrotic pulse is so frequently associated. Even with high fever the pulse may not be greatly accelerated. As the disease progresses the pulse becomes more rapid, feebler, and small. In the extreme prostration of severe cases it may reach 150 or more, and is a mere undulation—the so-called running pulse. The lowered arterial pressure is manifest in the dusky lividity of the skin and coldness of the hands and feet.

During convalescence the pulse gradually returns to normal, and occasionally becomes very slow. After no other acute fever do we so frequently meet with bradycardia. I have counted the pulse as low as thirty, and instances are on record of still fewer beats to the minute.

The *heart-sounds* are at first clear and loud, and free from murmur, but in severe cases, as the prostration develops, the first sound becomes feeble and there is often to be heard, at the apex and along the left sternal margin, a soft systolic murmur. The first sound may be gradually annihilated, as pointed out by Stokes. In the extreme feebleness of the ataxic forms, the first and second sound become very similar and the long pause is much shortened.

Of cardiac complications, *pericarditis* is rare and has been met with chiefly in children and in association with pneumonia. It was not present in any of my cases and occurred in only fourteen of the two thousand

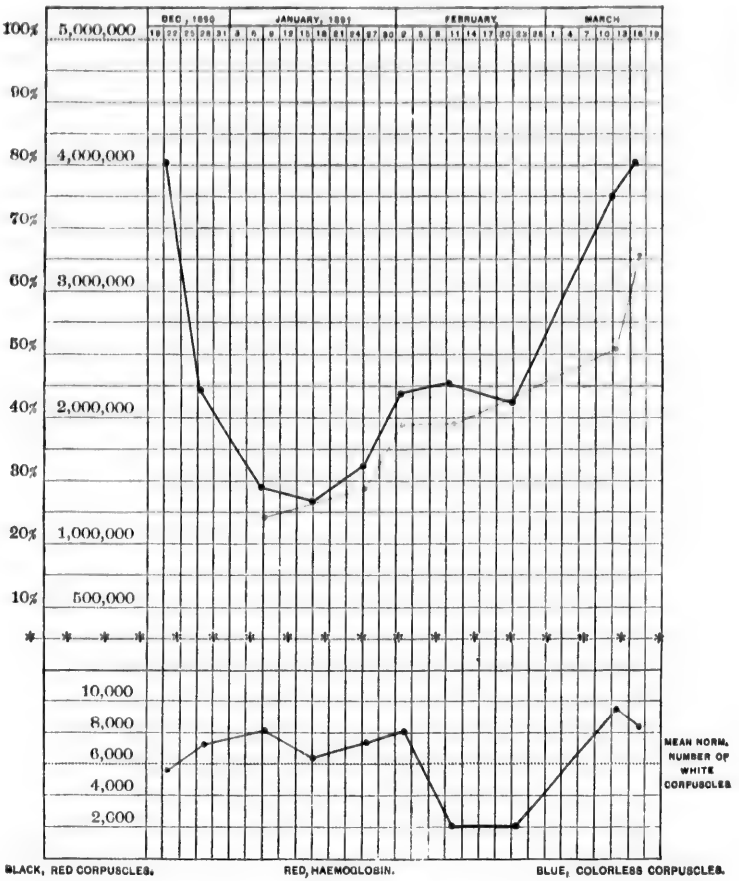

CHART III.

Munich post-mortems. *Endocarditis* is also uncommon. I saw one case at the Philadelphia Hospital. It must be very rare, as there were only eleven cases noted in the Munich records. Myocarditis is more common. The following statement may be made with reference to the condition of the heart-muscle in this disease: In protracted cases the muscle-fibre is usually soft, flabby, and of a pale yellowish-brown color. The softening may be extreme, though rarely of the grade described by Stokes, in which, when held apex up by the vessels, the organ collapsed over the hand,

forming a mushroom-like cap. Microscopically, the fibres may show little or no change, even when the impulse of the heart has been extremely feeble. A granular parenchymatous degeneration is common. Fatty degeneration may be present, particularly in long-standing cases with anæmia. The hyaline change is not common. The segmenting myocarditis, in which the cement substance is softened so that the muscle-cells separate, has also been found, but it is probably a post-mortem change.

Complications in the Arteries.—Obliteration of large or small arterial trunks is one of the rare complications of typhoid fever. A considerable number of cases are scattered through the literature. The obliteration may be due either to embolism or to thrombosis. In a majority of cases the femoral artery is involved and gangrene of the foot and leg occurs. In several cases there has been obliteration of both femorals with extension of the clot into the aorta and gangrene of both legs. In a case which I saw with Roddick, of Montreal, the obliteration of the left femoral occurred on the sixteenth day. On the twentieth day the patient had pain in the right leg and there was no pulsation in the femoral artery. Gangrene gradually developed in both feet, and death took place in the sixth week. In these cases the condition is probably due to thrombosis, not embolism, and is associated with a blood state which favors clotting, or possibly with a local arteritis. The condition is not invariably fatal. Of twenty cases collected by Barchoud,* eight died.

Thrombi in the Veins.—This is a much more frequent complication, and, according to Murchison, is met with in about one per cent of the cases. It occurs most frequently in a crural vein, and more commonly in the left than in the right; due possibly, as suggested by Liebermeister, to the fact that the left common iliac vein is crossed by the right iliac artery, and does not permit of so free a flow of blood as in the right vein. Thrombosis is indicated by enlargement and œdema of the limb, but gangrene never results from obstruction of the vein alone. It is not a very unfavorable complication. In one case of my series the thrombus had suppurated and there was pyæmia. Occasionally the thrombosis may extend into the pelvic veins and into the vena cava. In one instance the thrombus was in the right circumflex iliac vein alone, and the superficial veins on the right side of the abdomen were in consequence greatly enlarged. Sudden death has been caused by dislodgment of a thrombus.

Infarcts in the kidneys, spleen, and lungs are by no means uncommon in typhoid fever. They are associated usually with thrombosis in the arteries, rarely with embolism.

Digestive System.—Loss of appetite is early, and, as a rule, the relish for food is not regained until convalescence. Thirst is constant, and should be fully and freely gratified. Even when the mind becomes benumbed and the patient no longer asks for water, it should be freely given.

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* Paris Thesis, 1881.

The *tongue* presents the changes inevitable in a prolonged fever, but there are no distinctive characters. Early in the disease it is moist, swollen, and coated with a thin white fur, which, as the disease progresses, becomes denser. It may remain moist throughout. In severe cases, particularly those with delirium, the tongue becomes very dry, partly owing to the fact that such patients breathe with the mouth open. It may be covered with a brown or brownish-black fur, or with crusts between which are cracks and fissures. In these cases the teeth and lips may be covered with a dark brownish matter called *sordes*—a mixture of food, epithelial *débris*, and micro-organisms. By keeping the mouth and tongue clean from the outset the fissures, which are extremely painful, may be prevented. During convalescence the tongue gradually becomes clean, and the fur is thrown off, either insensibly or occasionally in flakes.

The secretion of saliva is often diminished; salivation is rare.

Parotitis is not so common as in typhus fever. It was present in forty-five of the two thousand Munich cases. It did not occur in any of my series of fatal cases. It is usually unilateral, and in a majority of cases goes on to suppuration. It is regarded as a very fatal complication, but recovery has followed in four or five of my cases. It undoubtedly may arise from extension of inflammation along Steno's duct. This is probably not so serious a form as when it arises from metastatic inflammation.

The *pharynx* may be the seat of slight catarrh. Sometimes the fauces are deeply congested. Membranous pharyngitis is a serious and fatal complication, which may come on in the third week.

The *gastric symptoms* are extremely variable. Nausea and vomiting are not common. There are instances, however, in which vomiting, resisting all measures, is a marked feature from the outset, and may directly cause death from exhaustion. Vomiting does not often occur in the second and third week, unless associated with some serious complication. In a few of these cases ulcers have been found in the stomach.

Of intestinal symptoms, *diarrhœa* is the most important. In some epidemics constipation exists, but in any long series of cases diarrhœa will be found to be a prominent feature of the disease. Its absence must not be taken as an indication that the intestinal disease is of slight extent. I have seen, on several occasions, the most extensive infiltration and ulceration of the Peyer's glands of the small intestine, with the colon filled with solid fæces. The diarrhœa is caused less by the ulcers than by the associated catarrh, and, as in tuberculosis, it is probable that when this is in the large intestine the discharges are more frequent. It is most common toward the end of the first and throughout the second week, but it may not occur until the third or even the fourth week. The number of discharges ranges from three to eight or ten in the twenty-four hours. They are usually abundant, thin, grayish-yellow, granular, of the consistency and appearance of pea-soup, and resemble very much, as Addison

remarked, the normal contents of the small bowel. The reaction is alkaline and the odor offensive. On standing, the discharges separate into a thin serous layer, containing albumen and salts, and a lower stratum, consisting of epithelial *débris*, remnants of food, and numerous crystals of triple phosphates. Blood may be in small amount, and only recognized

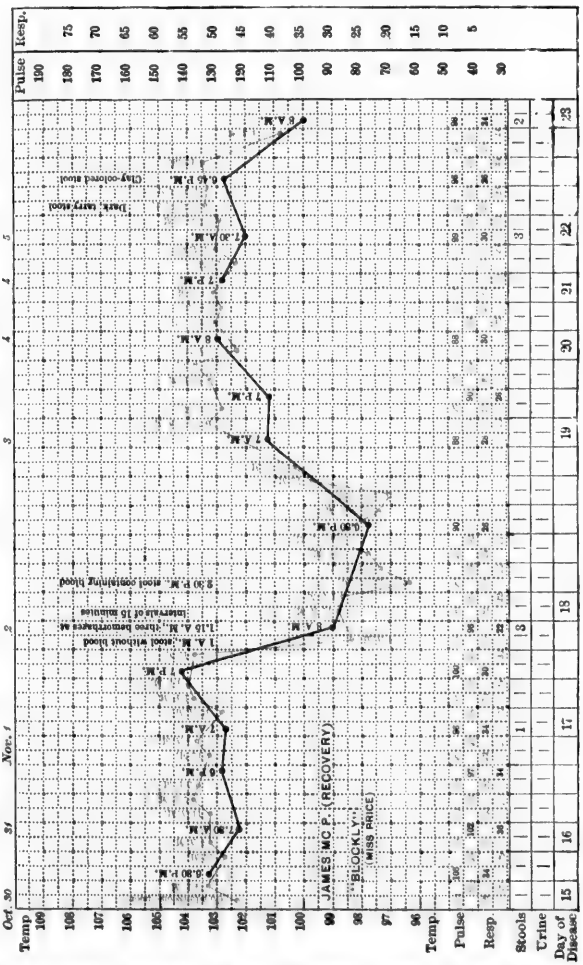


CHART IV.—Hæmorrhage from the bowels. Rapid fall of temperature.

by the microscope. Sloughs of the Peyer's glands occur either as grayish-yellow fragments or occasionally as ovoid masses, an inch or more in length, in which portions of the bowel tissue may be found.

Hæmorrhage from the bowels is a serious complication, occurring in from 3 to 5 per cent of all cases. It occurred in ninety-nine of the two

thousand Munich autopsies, and it was present in nine of my cases. There may be only a slight trace of blood in the stools, but too often it is a profuse, free hæmorrhage, which rapidly proves fatal. It occurs most commonly between the end of the second and the beginning of the fourth week, the time of the separation of the sloughs. Occasionally it results simply from the intense hyperæmia. It usually comes on without warning. A sensation of sinking or collapse is experienced by the patient, the temperature falls, and may, as in the annexed chart, drop eight or ten degrees in a few hours. Fatal collapse may supervene before the blood appears in the stool. Hæmorrhage usually occurs in cases of considerable severity. Graves and Trousseau held that this was not a very dangerous symptom, but statistics show that death follows in from thirty to fifty per cent of the cases.

It must not be forgotten that melæna may also be part of a general hæmorrhagic tendency, in which case it is associated with petechiæ and hæmaturia.

Meteorism is a frequent symptom, and if of moderate grade is not serious, but when excessive it is usually of ill-omen. Owing to defective tone in the walls, in severe cases owing to infiltration with serum, gas accumulates in the small and large bowels, particularly in the latter. It is rightly held to be to some extent a measure of the intensity of the local lesions. When extreme, it pushes up the diaphragm and interferes very much with the action of the heart and lungs. It undoubtedly also favors perforation.

Abdominal tenderness on pressure and gurgling in the right iliac fossa exist in a large proportion of all the cases. The tenderness may be more or less diffuse over the abdomen, but it is commonly limited to the right side It is rarely excessive and may be elicited only on deep pressure. Gurgling indicates simply the presence of gas and fluid fæces in the colon and cæcum.

Perforation of an ulcer into the peritonæum, the most serious abdominal complication of the disease, occurred in one hundred and fourteen of the two thousand Munich cases, and in fifteen of the sixty-four cases of my series It is usually indicated by the onset of sudden acute pain in the abdomen, and symptoms of collapse. It is most common at the end of the second or in the third week, but in one of my cases it occurred as early as the eighth day and in another in the sixth week, two weeks after the evening temperature had become normal. It is not infrequently associated with hæmorrhage. The presence of indigestible food, severe vomiting, excessive meteorism, and ascarides have been assigned as causes. This accident is much more common in men than in women. The perforation is usually in the ileum, but may occur in the colon. As a rule it promptly causes symptoms of peritonitis—distention of the abdomen, marked tenderness, rigidity of the abdominal walls, vomiting, a collapsed, pinched expression, and a rapid, small pulse. In very severe cases with marked

mental disturbance the symptoms may not excite suspicion, but the temperature usually falls and the symptoms of collapse are well marked. The diagnosis is easy, except in cases in which tympanites and tenderness have been prominent features, when it may be very difficult to say whether perforation has occurred. An indication of value in such instances is the obliteration of the liver dulness by gas in the peritoneal cavity, a symptom upon which Alonzo Clark and Flint laid great stress, and the value of which I have on several occasions been able to demonstrate. It is somewhat lessened by the fact that extreme tympany may almost, if not quite, obliterate the liver dulness. Recovery from perforation is undoubtedly possible, though rare.

Peritonitis without perforation may also occur by extension from the ulcer or occasionally by rupture of a softened mesenteric gland. It was present in 2·2 per cent of the Munich autopsies.

The *spleen* is invariably enlarged in typhoid fever, and in a majority of cases the edge can be felt below the costal margin. By the end of the first week the enlargement is evident, unless there is great distention of the colon, when the spleen may be pushed far back and difficult to feel. Even the normal area of dulness may not be obtainable. I have seen a very large spleen post mortem, when during life the increase in size was not observable. Toward the fourth week it diminishes in size. In four of my autopsies it weighed less than normal. Infarcts and abscesses are occasionally found. Rupture of the spleen in typhoid fever, due to a slight blow, has been seen by Bartholow. Spontaneous rupture may also occur.

Liver.—Symptoms on the part of this organ are rare. Enlargement is occasionlly detected. Jaundice is a very rare complication. It may be either of a catarrhal nature or due to parenchymatous changes. It was present in only 1·1 per cent of the Munich autopsies. Abscess of the liver is a very rare sequela.

Respiratory System.—*Epistaxis* is an early symptom in many cases, and precedes typhoid fever more commonly than it does any other febrile affection. It is occasionally profuse and serious.

Laryngitis is not very common. The ulcers and the perichondritis have already been described. Œdema apart from ulceration is rare. In this country the laryngeal complications of typhoid fever seem much less frequent than on the Continent. I have seen ulcers in only four or five instances, and twice only perichondritis, both of which cases recovered, one after the expectoration of large portions of the thyroid cartilage.

Bronchitis is one of the most frequent initial symptoms. It is indicated by the presence of numerous piping râles. It may come on with great severity, and in a case at the Philadelphia Hospital I regarded for several days the bronchial catarrh as the primary affection. The smaller tubes may be involved, producing urgent cough and even slight cyanosis. Collapse and lobular pneumonia may also occur.

Lobar pneumonia is met with under two conditions:

1. It may be the initial symptom of the disease. After an indisposition of a day or so, the patient is seized with a chill, has high fever, pain in the side, and within forty-eight hours there are signs of consolidation, and the evidences of an ordinary lobar pneumonia. The intestinal symptoms may not develop until toward the end of the first week or later; the pulmonary symptoms persist, crisis does not occur; the aspect of the patient changes, and by the end of the second week the clinical picture is that of typhoid fever. Spots may then be present and doubts as to the nature of the case are solved. In other instances, in the absence of a characteristic eruption the case remains dubious, and it is impossible to say whether the disease has been pneumonia, in which the so-called typhoid symptoms have developed, or whether it was typhoid fever with early implication of the lungs. Whether this condition depends upon the pneumococcus or is the result of an early localization of the typhoid bacillus has not yet been settled. I have twice performed autopsies in cases of this *pneumo-typhus*, as it is called by the French and Germans, and can speak positively of [illegible] with all the symptoms of a frank pneumonia.

2. Lobar pneumonia forms a serious and by no means infrequent complication of the second or third week. It was present in over 8 per cent of the Munich cases and occurred in nine of my cases. The symptoms are usually not marked There may be no rusty sputa, and, unless sought for, the condition is frequently overlooked. Infarction, abscess and gangrene are occasional pulmonary complications.

Hypostatic congestion of the lungs and œdema, due to enfeebled circulation in the later periods of the disease, are very common. The physical signs are defective resonance at the bases, feeble breath-sounds, and, on deep inspiration, moist râles. Pleurisy is by no means an uncommon complication. It was present in about 8 per cent of the Munich autopsies. It may develop slowly in convalescence, in which case it is almost always purulent. Another occasional pulmonary complication is *hæmoptysis*, which I once saw at the height of the disease. After death, no lesions of the lungs or bronchi were discovered. Miliary tuberculosis occasionally develops, and some writers hold that there is a greater susceptibility to infection with the tubercle bacillus after this than after other fevers.

Nervous System.—As already noted, the disease may set in with intense and persisting headache or an aggravated form of neuralgia. There are cases in which the effect of the poison is manifested on the nervous system early and with the greatest intensity. There are headache, photophobia, retraction of the neck, marked twitching of the muscles, rigidity, and even convulsions. In such cases the diagnosis of meningitis is invariably made. I have examined post mortem three such cases, in two of which the diagnosis of cerebro-spinal fever had been made. In not one of them was there any trace of meningeal inflammation, only the most intense congestion of the cerebral and spinal pia. Meningitis, however, may occur, but is extremely rare, as shown by the Munich record, in which

there were only eleven among the two thousand cases. Stokes's dictum that "there is no single nervous symptom which may not and does not occur independently of any appreciable lesion of the brain, nerves, or spinal cord," is too often forgotten.

Delirium is present in all severe cases. It is certainly less frequent under a rigid plan of hydrotherapy. It may be present from the outset, but usually does not develop until the second and sometimes not until the third week. It may be slight and only nocturnal. It is, as a rule, a quiet delirium, though there are cases in which the patient is very noisy and constantly tries to get out of bed, and, unless carefully watched, may escape. The patient does not often become maniacal. In heavy drinkers the delirium may have the character of delirium tremens. Even in cases which have no positive delirium, the mental processes are usually dulled and the patient is listless and apathetic. In severe cases the patient passes into a condition of unconsciousness. The eyes may be open, but he is oblivious to all surrounding circumstances and neither knows nor can indicate his wants. The urine and fæces are passed involuntarily. In this pseudo-wakeful state, or coma vigil as it is called, the eyes are open and the patient is constantly muttering. The lips and tongue are tremulous; there is twitching of the fingers and wrists — subsultus tendinum and carphologia. He picks at the bedclothes or grasps at invisible objects. These are among the most serious symptoms of the disease, and always indicate danger.

Among important complications and sequelæ are several nervous affections. The *paralyses* are due in the majority of instances to neuritis. It may be of a paraplegic type, or may involve only one or two nerves. Occasionally, as in a case reported by George Ross,* all four limbs are affected.

Possibly some of these cases are due to poliomyelitis, not to neuritis. This affection does not always follow, but may come on at the height of the disease, as in a case recently under my care, in which during the second week neuritis developed in both arms. Among other sequences may be mentioned aphasia, which is more apt to occur in young children, and great slowness of speech, which may or may not be associated with mental weakness.

Post-febrile *insanity* is perhaps more frequent after typhoid than after any other disease. Wood regards it as confusional insanity, the result of impaired nutrition and exhaustion of the nervous centres. Five cases have come under my observation, in four of which recovery took place.

Disturbances of the organs of the special senses are rare. Otitis media occasionally develops. Ocular symptoms are uncommon.

Renal System.—Retention of urine is an early symptom in many

* Paralysis in Typhoid Fever. Transactions of the Association of American Physicians, vol. iii.

cases, and is more frequent in some epidemics than in others. The urine is usually diminished at first, has the ordinary febrile characters, and the pigments are increased. Later in the disease it is more abundant and lighter in color.

Ehrlich has described a reaction, which he believes is rarely met with except in typhoid fever. This so-called *diazo-reaction* is produced as follows: Two solutions are employed, kept in separate bottles: one containing a saturated solution of sulphanilic acid in a solution of hydrochloric acid (50 c. c. to 1,000 c. c.); the other a ½ per cent solution of sodium nitrite. To make the test, a few cubic centimetres of urine are placed in a small test-tube with an equal quantity of a mixture of solution of the sulphanilic acid (40 c. c.) and the sodium nitrite (1 c. c.), the whole being thoroughly shaken. One cubic centimetre of ammonia is then allowed to flow carefully down the side of the tube, forming a colorless zone above the yellow urine, and at the junction of the two a deep brownish-red ring will be seen if the reaction is present. With normal urine a lighter brownish ring is produced, without a shade of red. The color of the foam of the mixed urine and reagent, and the tint they produce when largely diluted with water, are characteristic, being in both cases of a delicate rose-red if the diazo-reaction be present; but if not, brownish-yellow.

In twenty-six cases at my clinic, Simon found the reaction in twenty-two. It may be present previous to the occurrence of the rash, and as late as the twenty-second day. The value of the test is lessened by its occurrence in cases of miliary tuberculosis, and occasionally in the acute diseases associated with high fever.

The renal complications in typhoid fever may be thus grouped:

(*a*) Febrile albuminuria, which is very common and of no special significance; thus, in the first seventy-five cases admitted to the Johns Hopkins Hospital, albumen was present in forty-six, and in twenty-five cases casts were also found. In only two of these cases were there indications of an acute Bright's disease.

(*b*) Acute nephritis occurring at the onset or during the height of the disease—the *nephro-typhus* of the Germans, the *fièvre typhoïde à forme rénale* of the French—may set in, with all the symptoms of the most intense Bright's disease, masking in many instances the true nature of the malady. After an indisposition of a few days there may be fever, pain in the back, and the passage of a small amount of bloody urine. In a recent case* the early symptoms were all those of the most severe nephritis, and death occurred on the fourteenth day from perforation of the bowel. In other instances, as in a case reported in the same paper, the nephritis sets in at the end of the first or during the second week, and may modify con-

* Acute Nephritis in Typhoid Fever. Johns Hopkins Hospital Reports, February, 1890.

siderably the character of the disease, and even render the diagnosis doubtful.

(*c*) The nephritis of convalescence. This is more common but less serious. It develops after the fall of the fever, and is usually associated with œdema. It does not present characters different from the ordinary post-febrile nephritis.

(*d*) The remarkable lymphomatous nephritis described by E. Wagner and others, and already referred to in the section on morbid anatomy, produces, as a rule, no symptoms.

(*e*) *Post-typhoid pyelitis.*—In this the pelves of the kidney and the calices are at first covered with a membranous exudation, but erosion and ulceration may subsequently occur. There may be blood and pus in the urine. This condition occurred in three of my cases, in one of which it was associated with extensive membranous inflammation of the bladder.

Simple catarrh of the bladder is rare.

Orchitis is occasionally met with during convalescence. Sadrain collected sixteen cases in the literature. It is usually associated with a catarrhal urethritis. Induration or atrophy may occur, and more rarely suppuration.

Osseous System.—A multiple arthritis occasionally occurs; more commonly it is limited to a single joint, and may pass on to suppuration. Spontaneous luxation may develop. Necrosis is not uncommon during convalescence. Keen collected thirty-seven cases after typhoid fever. It is probably always the result of a secondary infection. Its most usual seat is the tibia.

The *muscles* show in some cases the degeneration already referred to, but it does not cause any symptoms. Hæmorrhage occasionally occurs into the muscles, and late in the disease abscess may develop.

Association of other Diseases.—Erysipelas is a rare complication, most commonly met with during convalescence. In 1,420 cases at Basle it occurred ten times. Griesinger states that it is met with in 2 per cent.

Measles may develop during the fever or in convalescence. Chickenpox and noma have been reported in children. Pseudo-membranous inflammations may occur in the pharynx, larynx, or genitals. Malarial and typhoid fevers may be associated, but a majority of the cases of so-called typho-malarial fever are either remittent or true typhoid.

Varieties of Typhoid.—Typhoid fever is an extremely complex disease. Many forms have been described, some of which present exaggeration of common symptoms, others modification in the course, others again greater intensity of action on certain organs. As we have seen, when the nervous system is specially involved, it has been called the cerebro-spinal form; when the kidneys are early and severely affected, nephro-typhoid; when the disease begins with pulmonary symptoms, pneumo-typhoid; when the disease is characterized throughout by profuse

sweats, the sudoral form of the disease. It is a mistake, I think, to recognize or speak of these as varieties. It is enough to remember that typhoid may set in occasionally with symptoms localised in certain organs, and that many of its symptoms are extremely inconstant—in one epidemic uniform and text-book-like, in another slight or not met with. This diversified symptomatology has led to many clinical errors, and in the absence of the salutary lessons of morbid anatomy it is not surprising that practitioners have so often been led astray. We may recognize, with Murchison, the following varieties:

1. The *mild* and *abortive* forms. It is very important for the practitioner to recognize the mild type of typhoid fever, often spoken of as gastric fever or even regarded as simple febricula. In this form, the typhus *levissimus* of Griesinger, the symptoms are similar in kind but altogether less intense than in the graver attacks, although the onset may be sudden and severe. The temperature rarely reaches 103°, and the fever of onset may not show the gradual ascending evening record. The spleen is enlarged, the rose-spots may be marked; often they are very few in number. The diarrhœa is variable, sometimes it is not present. In such cases the symptoms may persist for from sixteen to twenty days.

In the abortive form the symptoms of onset may be marked with shivering and fever of 103° or even higher. The date of onset is often definite, a point upon which Jürgensen lays great stress. Rose-spots may occur from the second to the fifth day. Early in the second week or at the end of the first week the fever falls, often with profuse sweating, and convalescence is established. In this abortive form relapse may occur and may occasionally prove severe. When typhoid fever prevails extensively these cases are not uncommon. I agree with J. C. Wilson, who states that they are not nearly so common in this country as in Europe.

2. The *grave* form is usually characterized by high fever and pronounced nervous symptoms. In this category, too, come the very severe cases setting in with pneumonia and Bright's disease, and with the very intense gastro-intestinal or cerebro-spinal symptoms.

3. The *latent* or *ambulatory* form of typhoid fever, which is particularly common in hospital practice. The symptoms are often very slight, and the patient scarcely feels ill enough to go to bed. He has languor, perhaps slight diarrhœa, but keeps about and may even attend to his work throughout the entire attack. In other instances delirium sets in. The worst cases of this form are seen in sailors, who keep up and about, though feeling ill and feverish. When brought to the hospital they often develop symptoms of a most severe type of the disease. Hæmorrhage or perforation may be the first symptom of this ambulatory type. Sir W. Jenner has called attention to the dangers of this form, and particularly to the grave prognosis in the case of persons who have travelled far with the disease in progress.

There is a rare and fatal form of typhoid fever, characterized by cutaneous and mucous hæmorrhages.

An *afebrile* typhoid fever is recognized by authors. Liebermeister says that the cases were not uncommon at Basle. The patients presented lassitude, depression, headache, furred tongue, loss of appetite, slow pulse, and even the spots and enlarged spleen. I have no personal knowledge of such cases.

Typhoid Fever in Children.—Epistaxis rarely occurs; the rise in temperature is less gradual; the initial bronchial catarrh is often observed. The nervous symptoms are often prominent; there are wakefulness and delirium; diarrhœa is often absent. The rash may be very slight, but the most copious eruption I have ever seen was in a child of eight. Oddly enough, considering the readiness with which the lymph elements of the intestine in children are involved, the abdominal symptoms are slight. Fatal hæmorrhage and perforation are rare. Among the sequelæ, aphasia and bone lesions may be mentioned as more common in children than in adults. The mortality of typhoid fever in children is low. Forcheheimer, in the Cincinnati epidemic in 1888, treated seventy cases without a death.

Typhoid Fever in the Aged.—After the fortieth year the disease runs a less favorable course, and the mortality is very high. Of sixty-four fatal cases, seven were over forty years of age; one was aged sixty-three, another seventy. The fever is not so high, but complications are more common, particularly pneumonia and heart failure.

Relapse.—Relapses vary in frequency in different epidemics, and, it appears, in different places. The percentages of different authors range from 3 per cent (Murchison), 11 per cent (Bäumler) to 15 or 18 per cent (Immermann). In Wagner's clinic, from 1882 to 1886, there were 49 relapses in 561 cases. F. C. Shattuck reports 21 relapses in 129 cases. R. L. MacDonnell 1 relapse in 100 cases. A relapse is a repetition, sometimes only a summary, of the original attack. Von Ziemssen insists correctly that two of the three important symptoms—step-like temperature at onset, roseola, and enlarged spleen—should be present to determine the diagnosis of a relapse. The intestinal lesions are repeated, though with less intensity and regularity. It is to be carefully distinguished from the fever of convalescence—or recrudescence—which has already been described. This is usually transitory, not lasting longer than a day or two. There are occasional instances in which the fever lasts for four or five days without rose-spots, or without enlargement of the spleen, and it may be impossible to determine whether there has been a relapse or not. The true relapse usually sets in after complete defervescence. Irvine noted the average duration of the interval in his cases at a little over five days. In eleven of Shattuck's cases the relapse began before complete defervescence. The onset is usually abrupt, though the step-like ascent is sometimes well seen, as in Chart I. The

eruption may be seen as early as the third or fourth day. The attack is usually less severe and of shorter duration. Of Murchison's fifty-three cases the mean duration of the first attack was about twenty-six, of the interval eleven, of the relapse fifteen days. The mortality of the relapse is not high. The relapse may be repeated, and a third and fourth relapse may occur.

The relapse is a reinfection from within, but we are still quite ignorant of the conditions favoring its occurrence. It is not at all likely that any special methods of treatment favor the relapse, though hydrotherapy has labored under this reproach.

Diagnosis.—If the patient is seen from the outset there is rarely any difficulty in diagnosing typhoid fever of typical course. In the prefebrile period the headache, weakness, loss of appetite and epistaxis are extremely suggestive, and, with an ascending pyrexia, scarcely need the distinctive rash to clinch the diagnosis.

The early and intense localisation of the symptoms in certain organs is a frequent source of error in diagnosis.

Cases coming on with severe headache, photophobia, delirium, twitching of the muscles and retraction of the head are almost invariably regarded as cerebro-spinal meningitis. Under such circumstances it may for a few days be impossible to make a satisfactory diagnosis. I have thrice performed autopsies on cases of this kind in which no suspicion of typhoid fever had been present; the intense cerebro-spinal manifestations having dominated the scene. Until the appearance of abdominal symptoms or the rash, it may be quite impossible to determine the nature of the case. Cerebro-spinal meningitis is, however, a rare disease; typhoid fever a very common one, and the onset with severe nervous symptoms is by no means infrequent. Fully one half of the cases of the so-called brain-fever belong to this category.

I have already spoken of the misleading pulmonary symptoms, which occasionally develop at the very outset of the disease. The bronchitis rarely causes error, though it may be intense and attract the chief attention. More difficult are the cases setting in with chill and followed rapidly by pneumonia. I have brought such a case before the class one week as typical pneumonia, and a fortnight later shown the same case as undoubtedly one of typhoid fever. In another case, in which the onset was with definite pneumonia, no spots developed, and, though there were diarrhœa, meteorism, and the most pronounced nervous symptoms, the doubt still remains whether it was a case of typhoid fever or one of pneumonia in which severe secondary symptoms developed. There is less danger of mistaking the pneumonia which develops at the height of the disease, and yet this is possible, as in a case admitted a few months ago to my wards—a man aged seventy, insensible, with a dry tongue, tremor, ecchymoses upon the wrists and ankles, no rose-spots, enlargement of the spleen, and consolidation of his right lower lobe. It was very

natural, particularly since there was no history, to regard such a case as senile pneumonia with profound constitutional disturbance, but the autopsy showed the characteristic lesions of typhoid fever.

In malarial regions typhoid and remittent fevers are very frequently confounded. I confess myself unable to differentiate certain cases of malarial remittent from typhoid fever, without the blood examination. I have repeatedly, both in Philadelphia and Baltimore, sent cases to the wards as typhoid fever which subsequently proved to be ordinary malarial remittent. The patient comes with a history of *malaise*, weakness, diarrhœa, perhaps vomiting; the tongue is furred and white, the cheeks are flushed, the spleen is slightly enlarged, temperature 102° or 103°. There may indeed be delirium, and the clinical picture of the early stage of typhoid fever may be complete. On at least two occasions I have shown such cases to my class as typhoid fever, and several times patients have been sent to the wards with instructions to have the head shaved and to begin the baths. The only safeguard against error is the examination of the blood, which should be done systematically in regions in which malaria prevails. The presence of Laveran's organisms is distinctive and absolutely diagnostic.

Acute miliary tuberculosis is not infrequently mistaken for typhoid fever. The points in differential diagnosis will be discussed under that disease. Tuberculous peritonitis in certain of its forms may closely simulate typhoid fever.

Puncture of the spleen for the purpose of obtaining cultures is justifiable only in exceptional circumstances.

Prognosis.—The mortality ranges from 10 to 30 per cent. Of the enormous number of deaths analyzed by Murchison, the mortality was nearly 19 per cent. The death-rate at the Montreal General Hospital, for twenty years, was 11·2 per cent. In recent years the mortality in typhoid fever has certainly diminished, and, under the influence of Brand, the reintroduction of hydrotherapy has reduced the mortality in institutions in a remarkable manner, even as low as 5 or 6 per cent. Especially unfavorable symptoms are high fever, delirium with toxic symptoms, hæmorrhage—though by some this is not thought very unfavorable—and peritonitis.

Sudden Death.—It is difficult in many cases to explain this most lamentable of accidents in the disease. There are cases in which neither cerebral, renal, nor cardiac changes have been found, and instances too in which it does not seem likely that there could have been a special localisation of the toxic poisons in the pneumogastric centres. McPhedran, in reporting a case of the kind, in which the post-mortem showed no adequate cause of death, suggests that the experiments of McWilliams on sudden cardiac failure probably explain the occurrence of death in certain of these cases in which neither embolism nor uræmia is present. Under conditions of abnormal nutrition there is sometimes induced a state

of *delirium cordis*, which may develop spontaneously, or, in the case of animals, on slight irritation of the heart, with the result of extreme irregularity and finally failure of action. It occurs more frequently in men than in women, according to Dewèvre's statistics, in a proportion of 114 to 26. It may occur at the height of the fever, and, as pointed out by Graves, may also happen during convalescence.

Fat subjects stand typhoid fever badly. The mortality in women is greater than in men. The complications and dangers are more serious in the ambulatory form in which the patient has kept about for a week or ten days. Early involvement of the nervous system is a bad indication; and the low, muttering delirium with tremor means a close fight for life. Prognostic signs from the fever alone are deceptive. A temperature above 104° may be well borne for many days if the nervous system is not involved.

Prophylaxis.—In cities the prevalence of typhoid fever is directly proportionate to the inefficiency of the drainage and the water-supply. There is no truer indication of the sanitary condition of a town than the returns of the number of cases of this disease. With the improvement in drainage the mortality in many cities has been reduced one half or even more. One of the most striking instances is afforded by the city of Munich. Von Ziemssen has published charts illustrating the extraordinary reduction in the prevalence of typhoid fever since the completion of the drainage system of that city. The average yearly number of admissions to hospital of cases of typhoid fever was, between the years 1866 and 188(594, while from 1881 to 1888 inclusive, the average has been only abou 100. During this same period the typhoid mortality of the whole city presented a yearly average of 208, but from 1881 to 1888 the yearly average was only 40.

By most rigid methods of disinfection much may be done to prevent the spread of the infection.

The following procedures, suggested by Fitz, should be carried out in hospital practice, and, with modifications, in private houses:

1. "Mattresses and pillows (when liable to become soiled) are to be protected by close-fitting rubber covers.

2. "Bed and body linen are to be changed daily. Bed-spreads, blankets, rubber sheets and rubber covers are to be changed at once when soiled. Avoid shaking any of the articles.

3. "All changed linens, bath-towels, rubber sheets and covers are to be immediately wrapped in a sheet soaked in carbolic acid (one to forty). Remove them to the rinse-house as soon as possible, and soak six hours in carbolic acid (one to forty). Then boil the linen for a half-hour, and wash with soft soap. The rubber sheets and covers are to be rinsed in cold water, dried, and aired for eight hours. The bed-spreads and blankets are to be aired eight hours daily.

4. "Feeding-utensils, immediately after using, are to be thoroughly cleansed in boiling water.

5. "Dejections are to be received into a bed-pan containing half a pint of carbolic acid (one to twenty). The nates are to be cleansed with paper, and afterward with a compress cloth wet with carbolic acid (one to forty).

6. "Add two quarts of carbolic acid (one to twenty), in divided portions, to the contents of the bed-pan; mix thoroughly by shaking and throw the liquid into the hopper. The bed-pan and hopper are to be cleansed with carbolic acid (one to twenty) and wiped dry. The cloth used for the above purpose is to be at once burned.

7. "The corpse is to be covered with a sheet wet with carbolic acid (one to forty).

8. "After the discharge of the patient from the hospital, the mattresses are to be aired every day for a week. The bedstead is to be washed with corrosive sublimate (one to one thousand).

9. "These directions are to be followed until the patient is free from fever."

When epidemics are prevalent the drinking-water and the milk used in families should be boiled. These precautions should be taken also by recent residents in any locality, and it is much safer for travellers to drink light wines or mineral water rather than ordinary water or milk.

Treatment.—(*a*) **General Management.**—The profession was long in learning that typhoid fever is not a disease to be treated by medicines. Careful nursing and a regulated diet are the essentials in a majority of the cases. The patient should be in a well-ventilated room (or in summer out of doors during the day), strictly confined to bed from the outset, and there remain until convalescence is well established. The bed should be single, not too high, and the mattress should not be too hard. The woven wire bed, with soft hair mattress, upon which are two folds of blanket, combines the two great qualities of a sick-bed, smoothness and elasticity. A rubber cloth should be placed under the sheet. An intelligent nurse should be in charge. When this is impossible, the attending physician should write out specific instructions regarding diet, treatment of the discharges, and the bed-linen.

(*b*) **Diet.**—Those forms of food should be given which are digested with the greatest ease, and which leave behind the smallest amount of residue to form fæces. Milk is the most suitable food. If used alone, three pints at least may be given to an adult in twenty-four hours, always diluted with water, lime-water, or aërated waters. Partially peptonized milk, when not distasteful to the patient, is occasionally serviceable. The stools of a patient on a strictly milk diet should be examined from time to time, to see if the milk is entirely digested. Fever patients often receive more than they can utilize, in which case masses of curds are seen in the stools, or microscopically fat-corpuscles in extraordinary abundance. Under these circumstances it is best to substitute, for part of the milk, mutton or chicken broths, or beef-juice, or a clear *consommé*, all of which may be made

very palatable by the addition of fresh vegetable juices. Some patients will take whey or buttermilk when the ordinary milk is distasteful. Thin barley-gruel, well strained, is an excellent food for typhoid-fever patients. Eggs may be given, either beaten up in milk or, better still, in the form of albumen-water. This is prepared by straining the whites of eggs through a cloth and mixing them with an equal quantity of water. It may be flavored with lemon, and, if the patient is taking spirits, whisky or brandy is very conveniently given with this. Patients who are unable to take milk can subsist for a time on this alone.

The patient should be encouraged to drink water freely, which may be pleasantly cold. Iced tea, barley-water, or lemonade may also be given, and there is no objection to coffee or cocoa in moderate quantities. Fruits are not, as a rule, allowable, though the juice of lemon or orange may be given. Typhoid patients should be fed at stated intervals through the day. At night it depends upon the general condition of the patient whether he should be aroused from sleep, or not. In mild cases it is not well to disturb the patient. When there is stupor, however, the patient should be roused for food at the regular intervals night and day.

Alcohol is not necessary in all cases, but may be given when the weakness is marked, the fever high, and the pulse failing. In young healthy adults, without nervous symptoms and without very high fever, alcohol is not required; but in any case, when the heart-beat is feeble and the first sound becomes obscure, if there is a muttering delirium, subsultus tendinum and a dry tongue, brandy or whisky should be freely given. In such a case from eight to twelve ounces of brandy in the twenty-four hours is a moderate amount.

(*c*) **Treatment of the Fever.**—The persistent pyrexia is in itself a danger, but perhaps not the chief danger. Cases with high fever alone, without delirium or signs of involvement of the nervous system, are not nearly so serious as those cases in which, with a temperature of 104°, there are pronounced nervous symptoms. For the fever and its concomitants there is no treatment so efficacious as that by cold water, introduced at the end of the last century by Currie, of Liverpool, and of late years forced upon the profession by Brand, of Stettin. In institutions a rigid system of hydrotherapy should be carried out. At my clinic the following plan is followed: Every third hour, if the temperature is above 102·5°, the patient is placed in a bath (at 70° Fahr.), which is wheeled to the bedside. In this he remains from fifteen to twenty minutes, and is then taken out, wrapped in a dry sheet and covered with a light blanket. Enough water is used to cover the patient's body to the neck. The head is sponged during the bath, and, if there is much torpor, cold water is poured over it from a height of a foot or two. The rectal temperature is taken immediately after the bath, and again three quarters of an hour later. The patient often complains bitterly when in the bath, and shivering and blueness are almost a constant sequence. Food is usually given

with a stimulant after the bath. The only contra-indications are peritonitis and hæmorrhage. Neither bronchitis nor pneumonia are so regarded. It is not necessary to renew the water in the bath more than once in the twenty-four hours. The accompanying chart shows the number of

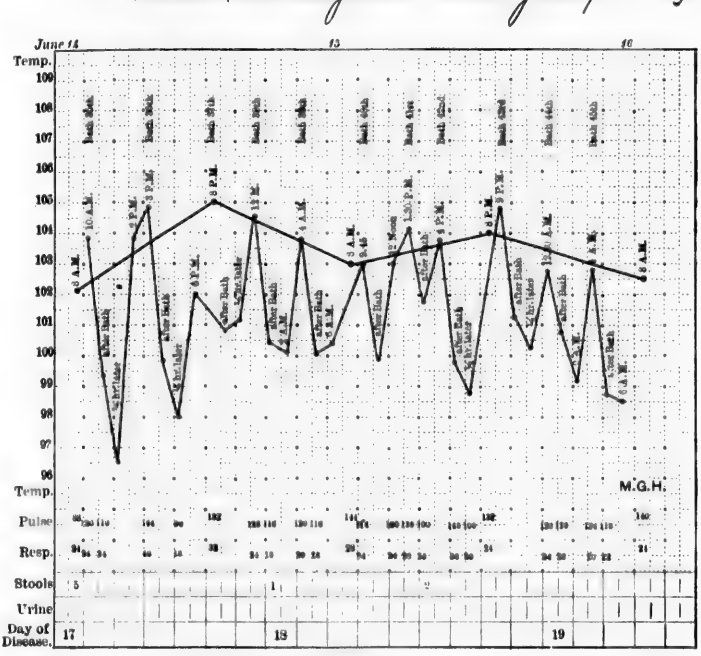


CHART V.

baths and the influence on the fever during two days of treatment. The good effects of the baths are: (1) the reduction of the fever; (2) the intellect becomes clearer, the stupor lessens, and the muscular twitchings disappear; (3) a general tonic action, particularly on the heart; (4) insomnia is lessened, the patient usually falling asleep for two or three hours after each bath; and (5), most important of all, the mortality is, under this plan of treatment, reduced to a minimum. This rigid method is not, however, without serious drawbacks, and personally I sympathize with those who designate it as entirely barbarous. To transfer a patient from a warm bed to a tub at 70° Fahr., and to keep him there twenty minutes or longer in spite of his piteous entreaties, does seem harsh treatment; and the subsequent shivering and blueness look distressing. A majority of our patients complain of it bitterly, and in private practice it is scarcely feasible.

The convincing statistics of the Brand method, as it is called, have

long been before the profession; but so far they have made but little impression in English-speaking communities. Cayley, of London, has been a warm advocate, but the rigid treatment is not often carried out in English or American institutions. J. C. Wilson, of Philadelphia, and Baruch, of New York, have pleaded for its general introduction into our hospitals. Among the most striking figures are those recently published by Hare, from the Brisbane Hospital, Australia. Under the expectant plan, 1,838 cases—mortality, 14·8 per cent; incomplete bath treatment, 171 cases—mortality, 12·3 per cent; strict bath treatment, 797 cases—mortality, 7 per cent.

The lukewarm bath, gradually cooled, is much more satisfactory in private practice. A bath at from 90° to 80°, and cooled down 10° or 12° by pouring cold water on the patient, will be found very satisfactory. When an insuperable objection to the bath exists, other hydrotherapeutic measures may be taken. The body may be sponged with tepid or cold water every time the temperature rises above 102·5°. If done thoroughly, taking limb by limb first, and then the trunk, occupying from twenty minutes to half an hour in the process, the rectal temperature may be reduced two or even three degrees. In private practice, when the bath is not available, the cold-pack is a good substitute. The patient is wrapped in a sheet wrung out of water at 60° or 65°, and cold water is sprinkled over him with an ordinary watering-pot. This is very efficacious in cases with pronounced nervous symptoms.

Medicinal antipyretics are rarely indicated. Quinine, which was employed so much in former years, has a slight though positive action, but its use has very wisely been restricted. The same may be said of the more recent antipyretics. Personally, I abandoned their employment some years ago. If given, antifebrin is the most suitable in doses of from four to eight grains. The action is prompt, and it is less depressing than antipyrin.

(*d*) **Antiseptic Medication.**—Very laudable endeavors have been made in many quarters to introduce methods of treatment directed toward the destruction of the typhoid bacilli, or the toxic agent which they produce, but so far without success. Good results have been claimed from the carbolic and iodine treatment. Others advocate corrosive sublimate or calomel, β-naphthol, and the salicin preparations. I can testify to the inefficiency of the carbolic acid and iodine and of the β-naphthol. With the mercurial preparations I have no experience. Fortunately for the patients, a majority of these medicines meet one of the two objects which Hippocrates says the physician should always have in view—they do no harm. Recently Burney Yeo has advocated the use of chlorine water and quinine as having a marked antiseptic action.

(*e*) **Treatment of the Special Symptoms.**—The abdominal pain and tympanites are best treated by fomentations or turpentine stupes. The latter, if well applied, give great relief. Sir William Jenner, at his clinic,

used to lay great stress on the advantages of a well-applied turpentine stupe. He directed it to be applied as follows: A flannel roller was placed beneath the patient, and then a double layer of thin flannel, wrung out of hot water, with a few drops of turpentine sprinkled upon it, was applied to the abdomen and covered with the ends of the roller.

The *meteorism* is a difficult and distressing symptom to treat. When the gas is in the large bowel, a tube may be passed or a turpentine enema given. For tympanites, with a dry tongue, turpentine was extensively used by the older Dublin physicians, and it was introduced into this country by the late George B. Wood. Unfortunately it is of very little service in the severer cases, which too often resist all treatment. The routine administration of turpentine in all cases of typhoid fever is a useless practice, for the perpetuation of which, in this generation, H. C. Wood is largely responsible. Stokes protested against it in his day, and very truly said that its use should be limited to the later periods of the disease, when it may sometimes be used with advantage, as Graves directs, in drachm doses every six hours. Sometimes, if beef-juice and albumen-water are substituted for milk, the distention lessens. Charcoal, bismuth, and β-naphthol may be tried.

For the *diarrhœa*, if severe—that is, if there are more than three or four stools daily—a starch and opium enema may be given; or, by the mouth, a combination of bismuth, in large doses, with Dover's powder; or the acid diarrhœa mixture, acetate of lead (grs. 2), dilute acetic acid (♏ 15–20), and acetate of morphia (gr. $\frac{1}{6}$–$\frac{1}{8}$). The stools should be examined to see that the diarrhœa is not aggravated by the presence of curds.

Constipation is present in many cases, and, though I have never seen it do harm, yet it is well every third or fourth day to give an ordinary enema. I have never used the initial dose of calomel, which is so highly recommended by some practitioners. If a laxative is needed during the course of the disease, the Hunyadi-janos or Friedrichshall water may be given.

Hæmorrhage from the bowels is best treated with full doses of acetate of lead and opium. As absolute rest is essential, the greatest care should be taken in the use of the bed-pan. It is perhaps better to allow the patient to pass the motions into the draw sheet. Ice may be freely given, and the amount of food should be restricted for eight or ten hours. If there is a tendency to collapse, stimulants should be given and, if necessary, hypodermic injections of ether. The patient may be spared the usual styptic mixtures with which he is so often drenched. Turpentine is warmly recommended by certain authors.

Peritonitis.—In a majority of the cases this is an inevitably fatal complication. The only hope lies in restriction of the inflammation. Cases have unquestionably recovered. Morphia should be given subcutaneously. If the peritonitis be due to perforation, the question of

laparotomy may be discussed. If perforation has occurred in the second or third week, it would be useless under the circumstances to attempt to stitch a slit in the intestine; if, on the other hand, it occurs during convalescence, it is only right to give the patient a chance, and the operation should be performed.

Progressive *heart-failure* is one of the most frequent and perhaps one of the most serious of the conditions which the physician has to combat. As in other specific affections, this is in part due to the prolonged action of the fever and in part is a toxic effect. Alcohol is here our mainstay and can be given freely. Strychnine is most useful and may be given hypodermically in full doses. Whether digitalis is indicated in the failing heart of fevers is not yet settled. Personally, I am by no means convinced that it does good. Hypodermic injections of ether may be resorted to, and are sometimes helpful in tiding the patient over a critical period.

The *nervous symptoms* of typhoid fever are best treated by hydrotherapy. One special advantage of this plan is that the restlessness is allayed, the delirium quieted, and sedatives are rarely needed. In the cases which set in early with severe headache, meningeal symptoms and high fever, the cold bath, or in private practice the cold-pack, should be employed. An ice-cap may be placed on the head, and if necessary morphia administered hypodermically. The practice, in such cases, of applying blisters to the nape of the neck and to the extremities is, to paraphrase Huxham's words, an *unwholesome severity*, which should long ago have been discarded by the profession. For the nocturnal restlessness, so distressing in some cases, Dover's powder should be given. As a rule, if a hypnotic is indicated, it is best to give opium in some form. Pulmonary complications should, if severe, receive appropriate treatment.

In protracted cases very special care should be taken to guard against *bed-sores*. Absolute cleanliness and careful drying of the parts after an evacuation should be enjoined. The patient should be turned from side to side and propped with pillows, and the back can then be sponged with spirits. On the first appearance of a sore, the water or air bed should be used.

(*f*) **The Management of Convalescence.**—With the fall of the temperature to normal in the evening, and the disappearance of the other symptoms, the patient enters upon a stage which is often more difficult to manage than the attack itself. Convalescents from typhoid fever frequently cause greater anxiety than patients in the attack. The question of food has to be met at once, as the patient develops a ravenous appetite and clamors for a fuller diet. My custom has been not to allow solid food until the temperature has been normal for ten days. This is, I think, a safe rule, leaning perhaps to the side of extreme caution; but after all with eggs, milk toast, milk puddings, and jellies, the patient can take a fairly varied diet. Many leading practitioners allow solid food to a patient so soon as he desires it. Peabody gives it on the disappearance of

the fever; the late Austin Flint was also in favor of giving solid food early; and Naunyn, at the Strasburg Medical Clinic, told me that this was his practice. I had an early lesson in this matter which I have never forgotten. A young lad in the Montreal General Hospital, in whose case I was much interested, passed through a tolerably sharp attack of typhoid fever. Two weeks after the evening temperature had been normal, and only a day or two before his intended discharge, he ate several mutton chops, and within twenty-four hours was in a state of collapse from perforation. A small transverse rent was found at the bottom of an ulcer which was in process of healing. It is not easy to say why solid food, particularly meats, should disagree, but in so many instances an indiscretion in diet is followed by slight fever, the so-called *febris carnis*, that it is in the best interests of the patient to restrict the diet for some time after the fever has fallen. An indiscretion in diet may indeed precipitate a relapse. The patient may be allowed to sit up for a short time about the end of the first week of convalescence, and the period may be prolonged with a gradual return of strength. He should move about slowly, and when the weather is favorable should be in the open air as much as possible. The patient should be guarded at this period against all unnecessary excitement. Emotional disturbance not infrequently is the cause of a recrudescence of the fever. Constipation is not uncommon in convalescence and is best treated by enemata. A protracted diarrhœa, which is usually due to ulceration in the colon, may retard recovery. In such cases the diet should be restricted to milk, and the patient should be confined to bed; large doses of bismuth and astringent injections will prove useful.

The recrudescence of the fever does not require special treatment. The treatment of the relapse is essentially that of the original attack.

Among the dangers of convalescence may be mentioned tuberculosis, which is said by Murchison to be more common after this than after any other fever. There are facts in the literature favoring this view, but it is a rare sequence in this country.

II. TYPHUS FEVER.

Definition.—An acute infectious disease characterised by sudden onset, a maculated rash, marked nervous symptoms, and a termination, usually by crisis, about the end of the second week.

Etiology.—The disease has long been known under the names of hospital fever, spotted fever, jail fever, camp fever, and ship fever. In Germany it is known as *exanthematic* typhus, in contradistinction to *abdominal* typhus.

Typhus is now a rare disease. Sporadic cases occur from time to time in the large centres of population, but epidemics are infrequent. In this

country during the past ten years there have been very few outbreaks. In New York in 1881–'82 seven hundred and thirty-five cases were admitted into the Riverside Hospital; in Philadelphia a small epidemic occurred in 1883 at the Philadelphia Hospital.

The special elements in the etiology of typhus are overcrowding and poverty. As Hirsch tersely puts it, "Die Geschichte des Typhus ist die des menschlichen Elends." Overcrowding, lack of cleanliness, intemperance and bad food are predisposing causes. The disease still lurks in the worst quarters of London and Glasgow, and is seen occasionally in New York and Philadelphia. It is more common in Great Britain and Ireland than in other parts of Europe. Murchison held that the disease might originate spontaneously under favorable conditions. This opinion is suggested by the occurrence of local outbreaks under circumstances which render it difficult to explain its importation, but the analogy of other infectious diseases is directly against it. In 1877 there occurred a local outbreak of typhus at the House of Refuge, in Montreal, in which city the disease had not existed for many years. The overcrowding was so great in the basement-rooms of the refuge that at night there were not more than eighty-eight cubic feet of space to each person. Eleven persons were affected. It was not possible to trace the source of infection.

Typhus is one of the most highly contagious of febrile affections. In epidemics nurses and doctors in attendance upon the sick are almost invariably attacked. There is no disease which has so many victims in the profession. In the extensive epidemic in the early and middle part of this century many hundred physicians died in the discharge of their duty. Casual attendance upon cases in limited epidemics does not appear to be very risky, but when cases are aggregated together in wards the poison appears concentrated and the danger of infection is much enhanced. Bedding and clothes retain the poison for a long time.

The microbe of typhus fever has not yet been determined. Hlava found in twenty of thirty-three bodies, and twice during life, a strepto-bacillus, the relation of which to the disease has not yet been determined.

Morbid Anatomy.—The anatomical changes are those which result from intense fever. The blood is dark and fluid, the muscles are of a deep red color and often show a granular degeneration, particularly in the heart; the liver is enlarged and soft and may have a dull clay-like lustre; the kidneys are swollen; there is moderate enlargement of the spleen, and a general hyperplasia of the lymph-follicles. Peyer's glands are not ulcerated. Bronchial catarrh is usually, and hypostatic congestion of the lungs often, present. The skin shows the petechial rash.

Symptoms.—Incubation.—This is placed at about twelve days, but it may be less. There may be ill-defined feelings of discomfort. As a rule, however, the *invasion* is abrupt and marked by chills or a single rigor, followed by fever. The chills may recur during the first few days, and there is headache with pains in the back and legs. There is early pros-

tration, and the patient is glad to take to his bed at once. The temperature is high at first, and may attain its maximum on the second or third day. The pulse is full, rapid, and not so frequently dicrotic as in typhoid. The tongue is furred and white, and there is an early tendency to dryness. The face is flushed, the eyes are congested, the expression is dull and stupid. Vomiting may be a distressing symptom. In severe cases mental symptoms are present from the outset, either a mild febrile delirium or an excited, active, almost maniacal condition. Bronchial catarrh is common.

Stage of Eruption.—From the third to the fifth day the eruption appears—first upon the abdomen and upper part of the chest, and then upon the extremities and face; developing so rapidly that in two or three days it is all out. There are two elements in the eruption: a subcuticular mottling, "a fine, irregular, dusky red mottling, as if below the surface of the skin some little distance, and seen through a semi-opaque medium" (Buchanan); and distinct papular rose-spots which change to petechiæ. In some instances the petechial rash comes out with the rose-spots. Collie describes the rash as consisting of three parts —rose-colored spots which disappear on pressure, dark-red spots which are modified by pressure, and petechiæ upon which pressure produces no effect. In children the rash at first may present a striking resemblance to measles, and give as a whole a curiously mottled appearance to the skin. The term mulberry rash is sometimes applied to it. In mild cases the eruption is slight, but even then is largely petechial in character. As the rash is largely hæmorrhagic, it is permanent and does not disappear after death. Usually the skin is dry, so that sudaminal vesicles are not common. It is stated by some authors that a distinctive odor is present. During the second week the general symptoms are usually much aggravated. The prostration becomes more marked, the delirium more intense, and the fever rises. The patient lies on his back with a dull expressionless face, flushed cheeks, injected conjunctivæ, and contracted pupils. The pulse increases in frequency and is feebler, the face is dusky, and the condition becomes more serious. Retention of urine is common. Coma-vigil is frequent, a condition in which the patient lies with open eyes, but quite unconscious. Subsultus tendinum and picking at the bedclothes are frequently seen. The tongue is dry, brown, and cracked, and there are sordes on the teeth. Respiration is accelerated, the heart's action becomes more and more enfeebled, and death takes place from exhaustion. In favorable cases, about the end of the second week occurs the crisis, in which, often after a deep sleep, the patient awakes feeling much better and with a clear mind. The temperature falls, and although the prostration may be extreme, convalescence is rapid and relapse very rare. This abrupt termination by crisis is in striking contrast to the mode of termination in typhoid fever.

Fever.—The temperature rises steadily during the first four or five

days, and the morning remissions are not marked. The maximum temperature is usually reached by the fifth day, when the temperature may reach 105°, 106°, or 107°. In mild cases it seldom rises above 103°. After reaching its maximum the temperature generally continues with slight morning remissions until the twelfth or fourteenth day, when the crisis occurs, during which the temperature may fall below normal within twelve or twenty-four hours. Preceding a fatal termination, there is usually a rapid rise in the fever to 108° or even 109°.

The heart may early show signs of weakness. The first sound becomes feeble and almost inaudible, and a systolic murmur at the apex is not infrequent. Hypostatic congestion of the lungs occurs in all severe cases.

The brain symptoms are usually more pronounced than in typhoid, and the delirium is more constant.

The urine in typhus shows the usual febrile increase of urea and uric acid. The chlorides diminish or disappear. Albumen is present in a large proportion of the cases, but nephritis seldom occurs.

Variations in the course of the disease are naturally common. There are malignant cases which rapidly prove fatal within two or three days; the so-called *typhus siderans*. On the other hand, during epidemics there are extremely mild cases in which the fever is slight, the delirium absent, and convalescence is established by the tenth day.

Complications and Sequelæ.—Broncho-pneumonia is perhaps the most common complication. It may pass on to gangrene. In certain epidemics gangrene of the toes, the hands, or the nose, and in children noma or cancrum oris, have occurred. Meningitis is rare. Paralyses, which are probably due to the post-febrile neuritis, are not very uncommon. Septic processes, such as parotitis and abscesses in the subcutaneous tissues and in the joints, are occasionally met. Nephritis is rare. Hæmatemesis may occur.

Prognosis.—The mortality ranges in different epidemics from 12 to 20 per cent. It is very slight in the young. Children, who are quite as frequently attacked as adults, rarely die. After middle age the mortality is high, in some epidemics 50 per cent. Death usually occurs toward the close of the second week and is due to the toxæmia. In the third week it is more commonly due to pneumonia.

Diagnosis.—During an epidemic there is rarely any doubt, for the disease presents distinctive general characters. Isolated cases may be very difficult to distinguish from typhoid fever. While in typical instances the eruption in the two affections is very different, yet taken alone it may be deceptive, since in typhoid fever a roseolous rash may be abundant and there is occasionally a subcuticular mottling and even petechiæ. The difference in the onset, particularly in the temperature, is marked; but cases in which it is important to make an accurate diagnosis are not usually seen until the fourth or fifth day. The suddenness of the onset, the greater frequency of the chill, and the early prostration are the distinctive

features in typhus. The brain symptoms too are earlier. It is easy to put down on paper elaborate differential distinctions, which are practically useless at the bedside, particularly when the disease is not prevailing as an epidemic. In sporadic cases the diagnosis is sometimes extremely difficult. I have seen Murchison himself in doubt, and more than once I have known a diagnosis to be deferred until the *sectio cadaveris*. Severe cerebro-spinal fever may closely simulate typhus at the outset, but the diagnosis is usually clear within a few days. Malignant variola also has certain features in common with severe typhus, but the greater extent of the hæmorrhages and the bleeding from the mucous membranes make the diagnosis clear within a short time. The rash at first resembles that of measles, but in this disease the eruption is brighter red in color, often crescentic or irregular in arrangement, and appears first in the face.

The frequency with which other diseases are mistaken for typhus is shown by the fact that during and following the epidemic of 1881 in New York one hundred and eight cases were wrongly diagnosed—one eighth of the entire number—and sent to the Riverside Hospital (F. W. Chapin).

Treatment.—Practically the general management of the disease is like that of typhoid fever. Hydrotherapy should be thoroughly and systematically employed. Judging from the good results which we have obtained by this method in typhoid cases with nervous symptoms much may be expected from it. Certain authorities have spoken against it, but it should be given a more extended trial. Medicinal antipyretics are less suitable than in typhoid, as the tendency to heart-weakness is often more pronounced. As a rule the patients require from the outset a supporting treatment; water should be freely given, and alcohol in suitable doses according to the condition of the pulse.

The bowels may be kept open by mild aperients. The so-called specific medication, by sulphocarbolates, the sulphides, carbolic acid, etc., is not commended by those who have had the largest experience. The special nervous symptoms and the pulmonary symptoms should be dealt with as in typhoid fever. In epidemics, when the conditions of the climate are suitable, the cases are best treated in tents in the open air.

III. RELAPSING FEVER (*Febris recurrens*).

Definition.—A specific infectious disease caused by the spirochæte (spirillum) of Obermeier, characterised by definite febrile paroxysms which usually last six days and are followed by a remission of about the same length of time, then by a second paroxysm, which may be repeated three or even four times, whence the name relapsing fever.

Etiology.—This disease, which has also the names "famine fever" and "seven-day fever," has been known since the early part of the

eighteenth century, and has from time to time extensively prevailed in Europe and in Ireland. It is common in India, where the conditions for its development seem always to be present. The subject has been specially studied by Vandyke Carter, of Bombay. It was first seen in this country in 1844, when cases were admitted to the Philadelphia Hospital, which are described by Meredith Clymer in his work on fevers. Flint saw cases in 1850–'51. In 1869 it prevailed extensively in epidemic form in New York and Philadelphia; since then it has not appeared.

The special conditions under which it develops are very similar to those of typhus fever. Overcrowding and deficient food are the conditions which seem to promote the rapid spread of the virus. Neither age, sex, nor season seems to have any special influence. It is a contagious disease and may be communicated from person to person, but is not so contagious as typhus. Murchison thinks it may be transported by fomites. One attack does not confer immunity from subsequent attacks. In 1873 Obermeier described an organism in the blood which is now recognised as the specific agent. This spirillum, or more correctly spirochæte, is from three to six times the length of the diameter of a red blood-corpuscle, and forms a narrow spiral filament which is readily seen moving among the red corpuscles during a paroxysm. They are present in the blood only during the fever. Shortly before the crisis and in the intervals they are not found, though small glistening bodies, which are stated to be their spores, appear in the blood. The disease has been produced in human beings by inoculation of the blood during the paroxysm. It has also been produced in monkeys. Nothing is yet known with reference to the life history of the spirochæte.

Morbid Anatomy.—There are no characteristic anatomical appearances in relapsing fever. If death takes place during the paroxysm the spleen is large and soft, and the liver, kidneys and heart show cloudy swelling. There may be infarcts in the kidneys and spleen. The bone marrow has been found in a condition of hyperplasia. Ecchymoses are not uncommon.

Symptoms.—*Incubation* appears to be short, and in some instances the attack develops promptly after exposure; more frequently, however, from five to seven days elapse.

The *invasion* is abrupt, with chill, fever, and intense pain in the back and limbs. In young persons there may be nausea, vomiting, and convulsions. The temperature rises rapidly and may reach 104° on the evening of the first day. Sweats are common. The pulse is rapid, ranging from 110 to 130. There may be delirium if the fever is high. Swelling of the spleen can be detected early. Jaundice is common in some epidemics. The gastric symptoms may be severe. There are seldom intestinal symptoms. Cough may be present. Occasionally herpes is noted, and there may be miliary vesicles and petechiæ. During the paroxysm the blood invariably shows the spirochæte. After persisting with severity or even

with an increasing intensity for five or six days the crisis occurs. In the course of a few hours, accompanied by profuse sweating, sometimes by diarrhœa, the temperature falls to normal or even subnormal, and the period of apyrexia begins.

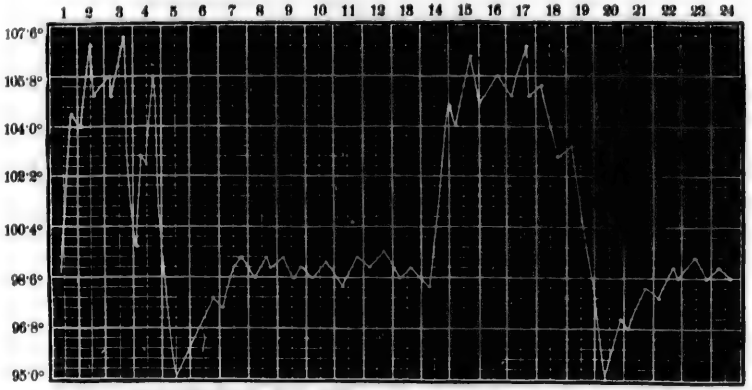


CHART VI.—Relapsing fever (Murchison).

The crisis may occur as early as the third day, or it may be delayed to the tenth; it usually comes, however, about the end of the first week. In delicate and elderly persons there may be collapse. The convalescence is rapid, and in a few days the patient is up and about. Then in a week, usually on the fourteenth day, he again has a rigor, or a series of chills; the fever returns and the attack is repeated. A second crisis occurs from the twentieth to the twenty-third day, and again the patient recovers rapidly. As a rule the relapse is shorter than the original attack. A second and a third may occur, and there are instances on record of even a fourth and a fifth. In epidemics there are cases terminating by crisis on the seventh or eighth day without the occurrence of relapse. In protracted cases the convalescence is very tedious, as the patient is much exhausted.

Relapsing fever is not a very fatal disease. Murchison states that the mortality is about 4 per cent. In the enfeebled and old, death may occur at the height of the original attack.

Complications are not frequent. In some epidemics nephritis and hæmaturia have occurred. Pneumonia appears to be frequent and may interrupt the typical course of the disease. The acute enlargement of the spleen may end in rupture, and the hæmorrhage from the stomach which has been met with occasionally is probably associated with this enlargement. Post-febrile paralyses may occur. Ophthalmia has followed certain epidemics, and may prove a very tedious and serious complication. Jaundice has already been mentioned. In pregnant women abortion usually takes place.

Diagnosis.—The onset and general symptoms may not at first be distinctive. At the beginning of an epidemic the cases are usually regarded as anomalous typhoid; but once the typical course is followed in a case the diagnosis is clear. The blood examination, which should be made in all doubtful cases of fever, affords a definite criterion by which the diagnosis can readily be made.

Treatment.—The paroxysm can neither be cut short nor its recurrence prevented. It might be thought that quinine, with its powerful action, would certainly meet the indications, but it does not seem to have the slightest influence. The disease must be treated like any other continued fever by careful nursing, a regular diet, and ordinary hygienic measures. Of special symptoms, pains in the back and in the limbs and joints demand opium. In enfeebled persons the collapse at the crisis may be serious, and stimulants with ammonia and digitalis should be given freely.

IV. SMALL-POX (*Variola*).

Definition.—An acute infectious disease characterised by an eruption which passes through the stages of papule, vesicle, pustule and crust. The mucous membranes in contact with the air may also be affected. Severe cases may be complicated with cutaneous and visceral hæmorrhages.

Etiology.—It has not yet been determined in what country small-pox originated. The disease is said to have existed in China many centuries before Christ. The *pesta magna* described by Galen (and of which Marcus Aurelius died) is believed to be small-pox. In the sixth century it prevailed, and subsequently, at the time of the Crusades, became widespread. It was brought to America by the Spaniards early in the sixteenth century. The first accurate account was given by Rhazes, an Arabian physician who lived in the ninth century, and whose admirable description is available in Greenhill's translation for the Sydenham Society. In the seventeenth century a thorough study of the disease was made by the illustrious Sydenham, who still remains one of the most trustworthy authorities on the subject.

Special events in the history of the disease are the introduction of inoculation into Europe, by Lady Mary Wortley Montagu, in 1718, and the discovery of vaccination by Jenner, in 1798.

Small-pox is one of the most virulent of contagious diseases, and persons exposed, if unprotected by vaccination, are almost invariably attacked. There are instances on record of persons insusceptible to the disease. It is said that Diemerbrock, a celebrated Utrecht professor in the seventeenth century, was not only himself exempt, but likewise many members of his family. One of the nurses in the small-pox department of the Montreal General Hospital stated that she had never been successfully vaccinated,

and she certainly had no mark. Such instances, however, of natural immunity are very rare.

Age.—Small-pox is common at all ages, but is particularly fatal to young children; thus, in the Montreal epidemic of 1885, 86 per cent of the deaths were of children under ten years of age. The *fœtus in utero* may be attacked, but only if the mother herself is the subject of the disease. The child may be born with the rash out or with the scars. More commonly the fœtus is not affected, and children born in a small-pox hospital, if vaccinated immediately, may escape the disease; usually, however, they die early.

Sex.—Males and females are equally affected.

Race.—Among aboriginal races small-pox is terribly fatal. When the disease was first introduced into America the Mexicans died by thousands, and the North American Indians have also been frequently decimated by this plague. It is stated that the negro is especially susceptible.

The Contagium develops in the system of the small-pox patient and is reproduced in the pustules. It exists in the secretions and excretions, and in the exhalations from the lungs and the skin. The dried scales constitute by far the most important element, and as a dust-like powder are distributed everywhere in the room during convalescence, becoming attached to clothing and various articles of furniture. The disease is probably contagious from a very early stage, though I think it has not yet been determined whether the contagion is active before the eruption develops. The poison is of unusual tenacity and clings to infected localities. It is conveyed by persons who have been in contact with the sick and by fomites. During epidemics it is no doubt widely spread in street-cars and public conveyances. It must not be forgotten that an unprotected person may contract a very virulent form of the disease from the mild varioloid.

The disease smoulders here and there in different localities, and when conditions are favorable becomes epidemic. Perhaps the most remarkable instance in modern times of the rapid extension of the disease occurred in Montreal in 1885. Small-pox had been prevalent in that city between 1870 and 1875, when it died out, in part owing to the exhaustion of suitable material and in part owing to the introduction of animal vaccination. The health reports show that the city was free from the disease until 1885. During these years vaccination, to which many of the French Canadians are opposed, was much neglected, so that a large unprotected population grew up in the city. On February 28th a Pullman-car conductor, who had travelled from Chicago, where the disease had been slightly prevalent, was admitted into the Hôtel-Dieu, the civic small-pox hospital being at the time closed. Isolation was not carried out, and on the 1st of April a servant in the hospital died of small-pox. Following her decease, with a negligence absolutely criminal, the authorities of the hospital dismissed all patients presenting no symptoms of contagion, who could go home. The

disease spread like fire in dry grass, and within nine months there died in the city, of small-pox, 3,164 persons.

The nature of the contagion of small-pox is still unknown. Weigert and others have described micro-organisms in the pock, but they are the ordinary pus cocci, and the part which they play in the affection is by no means certain. Still less definite are the observations on the occurrence of sporozoa in the pocks. It is not a little remarkable that in a disease which is rightly regarded as the type of all infectious maladies, the specific virus still remains unknown.

Morbid Anatomy.—A section of a papule as it is passing into the vesicular stage shows in the *rete mucosum*, close to the true skin, an area in which the cells are smooth, granular, and do not take the staining fluid. This represents a focus of coagulation-necrosis due, according to Weigert, to the presence of micrococci. Around this area there is active inflammatory reaction, and in the vesicular stage the rete mucosum presents reticuli, or spaces, which contain serum, leucocytes and fibrin filaments. The central depression or umbilication corresponds to the area of primary necrosis. In the stage of maturation the reticular spaces become filled with leucocytes and many of the cells of the rete mucosum become vesicular. The papillæ of the true skin below the pustule are swollen and infiltrated with embryonic cells to a variable degree. If the suppuration extends into this layer, scarring inevitably results; but if it is confined to the upper layer, it does not necessarily follow. In the hæmorrhagic cases, red corpuscles pass out in large numbers from the vessels and occupy the vesicular spaces. They infiltrate also the deeper layers of the epidermis in the skin adjacent to the papules. Frequently a hair-follicle passes through the centre of a papule.

In the mouth the pustules may be seen upon the tongue and the buccal mucosa, and on the palate. The eruption may be abundant also in the pharynx and the upper part of the œsophagus. In exceptionally rare cases the eruption extends down the œsophagus and even into the stomach. Swelling of the Peyer's follicles is not uncommon; the pustules have been seen in the rectum.

In the larynx the eruption may be associated with a fibrinous exudate and sometimes with œdema. Occasionally the inflammation passes deeply and involves the cartilages. In the trachea and bronchi there may be ulcerative erosions, but true pocks, such as are seen on the skin, do not occur. There are no special lesions of the lungs, but congestion and broncho-pneumonia are very common. The liver is sometimes fatty. A diffuse hepatitis, associated with intense congestion of the vessels and migration of the leucocytes, has been described; Weigert has noted small areas of necrosis.

There is nothing special in the condition of the blood, and even in the most malignant cases there are no microscopic alterations. In the blood-drop, however, it will be seen that the corpuscles, instead of forming

rouleaux, aggregate together in irregular clumps. The heart occasionally shows myocardial changes, parenchymatous and fatty; endocarditis and pericarditis are uncommon. French writers have described an endarteritis of the coronary vessels in connection with small-pox. The spleen is markedly enlarged. Apart from the cloudy swelling and areas of coagulation-necrosis, lesions of the kidneys are not common. Nephritis may occur during convalescence. Chiari has called attention to the frequency of orchitis in this disease. There are scattered areas of necrosis with cell infiltration.

In the hæmorrhagic form extravasations are found on the serous and mucous surfaces, in the parenchyma of organs, in the connective tissues, and about the nerve-sheaths. In one instance I found the entire retroperitoneal tissue infiltrated with a large coagulum, and there were also extensive extravasations in the course of the thoracic aorta. Hæmorrhages in the bone-marrow have also been described by Golgi. There may be hæmorrhages into the muscles. Ponfick has described the spleen as very firm and hard in hæmorrhagic small-pox, and such was the case in seven instances which I examined. The liver has been described as fatty in these rapid cases, but in five of my seven cases it was of normal size, dense, and firm. In two it was large and fatty; but one man had necrosis of the tibia, and the other was a drunkard. The ecchymoses are scattered over the meninges of the brain and cord, and in one case there was a clot in the right ventricle. In five of the cases there were areas of hæmorrhagic infarction of the lung. In four instances the pelves of the kidney were blocked with dark clots, which extended into the calices and down the ureters. In one instance the coats of the bladder were uniformly hæmorrhagic and not a trace of normal tissue could be seen. The extravasations in the mucous membrane of the stomach and intestines were numerous and large. Peyer's glands were swollen and prominent in four instances.

Symptoms.—Three forms of small-pox are described:

1. *Variola vera;* (*a*) Discrete, (*b*) Confluent.

2. *Variola hæmorrhagica;* (*a*) Purpura variolosa or black small-pox; (*b*) Hæmorrhagic pustular form, variola hæmorrhagica pustulosa.

3. *Varioloid*, or small-pox modified by vaccination.

1. **Variola Vera.**—The affection may be conveniently described under various stages: (*a*) *Incubation.* This is variously estimated at from seven to twelve days, or even longer. I have seen it develop on the eighth day after exposure to infection, and there are well-authenticated instances in which the stage of incubation has been prolonged to twenty days. It is unusual for patients to complain of any symptoms in this stage.

(*b*) *Invasion.*—In adults a chill and in children a convulsion are common initial symptoms. There may be repeated chills within the first twenty-four hours. Intense frontal headache, severe lumbar pains and vomiting are very constant features. The pains in the back and in the

limbs are more severe in the initial stage of this than of any other eruptive fever, and their combination with headache and vomiting is so sug-

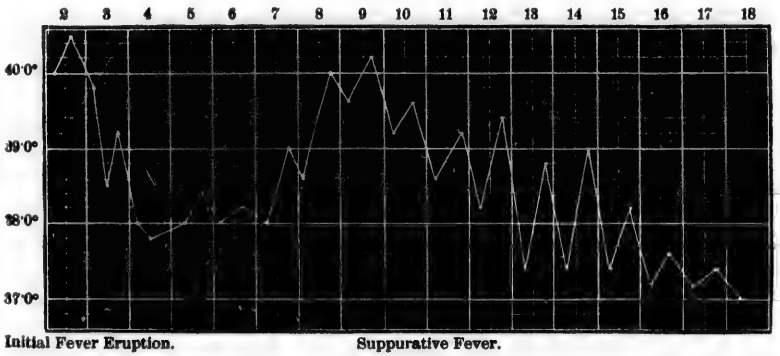


CHART VII.—True small-pox.

gestive that in epidemics precautionary measures may often be taken several days before the eruption decides positively the nature of the disease. The temperature rises quickly, and may on the first day be 103° or 104°. The pulse is rapid and full, not often dicrotic. In severe cases there may be marked delirium, particularly if the fever is high. The patient is restless and distressed, the face is flushed, and the eyes are bright and clear. The skin is usually dry, though occasionally there are profuse sweats. One cannot judge from these initial symptoms whether a case is likely to be discrete or confluent, as the most intense backache and fever may precede a very mild attack. Convulsions are not uncommon in children.

In this stage of invasion the so-called initial rashes may occur, of which two forms can be distinguished—the diffuse, scarlatinal, and the macular or measly form; either of which may be associated with petechiæ and occupy a variable extent of surface. In some instances they are general, but as a rule they are limited, as pointed out by Simon, either to the lower abdominal areas, to the inner surfaces of the thighs, and to the lateral thoracic region or to the axillæ. Occasionally they are found over the extensor surfaces, particularly in the neighborhood of the knees and elbows. These rashes, usually purpuric, are often associated with an erythematous or erysipelatous blush. The scarlatinal rash may come out as early as the second day and be as diffuse and vivid as in a true scarlatina. The measly rash may also be diffuse and identical in character with that of measles. Urticaria is only occasionally seen. It was present once in my Montreal cases. Apparently these initial rashes are more abundant in some epidemics than in others; thus they were certainly more numerous in the Montreal epidemics between 1870 and 1875 than they were in the more extensive epidemic in 1885. They occur in from 10 to 16 per cent

of cases. In the cases under my care in the small-pox department at the Montreal General Hospital the percentage was 13.* As will be subsequently mentioned these initial rashes have considerable diagnostic value.

(*c*) *Eruption.*—(1) In the *discrete form*, usually on the fourth day, small red spots appear on the forehead, particularly at the junction with the hair, and on the wrists. Within the first twenty-four hours from their appearance they occur on other parts of the face and on the extremities, and a few are seen on the trunk. As the rash comes out the temperature falls, the general symptoms subside, and the patient feels comfortable. On the fifth or sixth day the papules change into vesicles with clear summits. Each one is elevated, circular, and presents a little depression in the centre, the so-called umbilication. About the eighth day the vesicles change into pustules, the umbilication disappears, the flat top assumes a globular form and becomes grayish yellow in color, owing to the contained pus. There is an areola of injection about the pustules and the skin between them is swollen. This maturation first takes place on the face, and follows the order of the appearance of the eruption. The temperature now rises—secondary fever—and the general symptoms return. The swelling about the pustules is attended with a good deal of tension and pain in the face; the eyelids become swollen and closed. In the discrete form the temperature of maturation does not usually remain high for more than twenty-four or twenty-six hours, so that on the tenth or eleventh day the fever disappears and the stage of convalescence begins. The pustules rapidly dry, first on the face and then on the other parts, and by the fourteenth or fifteenth day desquamation may be far advanced on the face. There may be in addition vesicles in the mouth, pharynx, and larynx, causing soreness and swelling in these parts, with loss of voice. Whether pitting takes place depends a good deal upon the severity of the disease. In a majority of cases Sydenham's statement holds good, that "it is very rarely the case that the distinct small-pox leaves its mark."

(2) *The Confluent Form.*—With the same initial symptoms, though usually of greater severity, the rash appears on the fourth, or, according to Sydenham, on the third day. The more the eruption shows itself before the fourth day, the more sure it is to become confluent (Sydenham). The papules at first may be isolated and it is only later in the stage of maturation that the eruption is confluent. But in severer cases the skin is swollen and hyperæmic and the papules are very close together. On the feet and hands, too, the papules are thickly set; more scattered on the limbs; and quite discrete on the trunk. With the appearance of the eruption the symptoms subside and the fever remits, but not to the same extent as in the discrete form. Occasionally the temperature falls to normal and the patient may be very comfortable. Then, usually on the eighth day, the temperature again rises, the vesicles begin to change to

*The Initial Rashes of Small-pox. Canada Medical and Surgical Journal, 1875.

pustules, the hyperæmia about them becomes intense, the swelling of the face and hands increases, and by the tenth day the pustules have fully maturated, many of them have coalesced and the entire skin of the head and extremities is a superficial abscess. The fever rises to 103° or 104°, the pulse is from 110 to 120, and there is often delirium. As pointed out by Sydenham, salivation in adults and diarrhœa in children are common symptoms of this stage. There is usually much thirst. The eruption may also be present in the mouth, and usually the pharynx and larynx are involved and the voice is husky. Great swelling of the cervical lymphatic glands occurs. At this stage the patient presents a terrible picture, unequalled in any other disease; one which fully justifies the horror and fright with which small-pox is associated in the public mind. Even when the rash is confluent on the face, hands, and feet, the pustules remain discrete on the trunk. The danger, as pointed out by Sydenham, is in proportion to the number upon the face. "If upon the face they are as thick as sand it is no advantage to have them few and far between on the rest of the body." In fatal cases, by the tenth or eleventh day the pulse gets feebler and more rapid, the delirium is marked, there is subsultus, sometimes diarrhœa, and with these symptoms the patient dies. In other instances between the eighth and eleventh day hæmorrhagic symptoms develop. When recovery takes place, the patient enters on the eleventh or twelfth day the period of—

(*d*) *Desiccation.*—The pustules break and the pus exudes and forms crusts. Throughout the third week the desiccation proceeds and in cases of moderate severity the secondary fever subsides; but in others it may persist until the fourth week. The crusts in confluent small-pox adhere for a long time and the process of scarring may take three or four weeks. The crusts on the face fall off, but the tough epidermis of the hands and feet may be shed entire. We had in the small-pox department of the Montreal General Hospital several moulds in epithelium of the hands and feet.

2. **Hæmorrhagic small-pox** occurs in two forms. In one the special symptoms appear early and death follows in from two to six days. This is the so-called petechial or black small-pox—*purpura variolosa.* In the other form the case progresses as one of ordinary variola, and it is not until the vesicular or pustular stage that hæmorrhage takes place into the pocks or from the mucous membranes. This is sometimes called *variola hæmorrhagica pustulosa.*

Hæmorrhagic small-pox is more common in some epidemics than in others. It is less frequent in children than in adults. Of twenty-seven cases admitted to the small-pox department of the Montreal General Hospital there were three under ten years, four between fifteen and twenty, nine between twenty and twenty-five, seven between twenty-five and thirty-five, three between thirty-five and forty-five, and one above fifty. Young and vigorous persons seem more liable to this form. Several of my cases were above the average in muscular development. Men are more fre-

quently affected than women; thus in my list there were twenty-one males and only six females. The influence of vaccination is shown in the fact that of the cases fourteen were unvaccinated, while not one of the thirteen who had scars had been revaccinated.

The clinical features of the forms of hæmorrhagic small-pox are somewhat different.

In *purpura variolosa* the illness starts with the usual symptoms, but with more intense constitutional disturbance. On the evening of the second or on the third day there is a diffuse hyperæmic rash, particularly in the groins, with small punctiform hæmorrhages. The rash extends, becomes more distinctly hæmorrhagic, and the spots increase in size. Ecchymoses appear on the conjunctivæ, and as early as the third day there may be hæmorrhages from the mucous membranes. Death may take place before the rash appears. This is truly a terrible affection and well developed cases present a frightful appearance. The skin may have a uniformly purplish hue and the unfortunate victim may even look plum-colored. The face is swollen and large conjunctival hæmorrhages with the deeply sunken corneæ give a ghastly appearance to the features.

The mind may remain clear to the end. Death occurs from the third to the sixth day; thus in thirteen of my cases death took place on or before this date. The earliest death was on the third day and there were no traces of papules. There may be no mucous hæmorrhages; thus in one case of a most virulent character death occurred without bleeding early on the fourth day. Hæmaturia is perhaps most common, next hæmatemesis, and melæna was noticed in a third of the cases. Metrorrhagia was noticed in one only of the six females on my list. Hæmoptysis occurred in five cases. The pulse in this form of small-pox is rapid and often hard and small. The respirations are greatly increased in frequency and out of all proportion to the intensity of the fever. In the case of a negro, whose respirations the morning after admission were 32 and temperature 101°, after examining the lungs and finding nothing to account for the increased breathing, my suspicions were aroused, and even on the dark skin I was able on careful inspection to detect hæmorrhages in and about the papules.

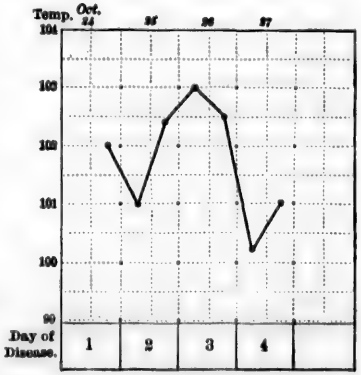


CHART VIII.—Hæmorrhagic small-pox.

The annexed chart is from a case of malignant small-pox which came on abruptly on Thursday, October 24, 1874, and which terminated early on the fourth day. It shows the moderate temperature range.

In *variola pustulosa hæmorrhagica* the disease progresses as an ordinary case of severe variola, and the hæmorrhages do not develop until the vesicular or pustular stage. The earlier the hæmorrhage the greater is the danger. There are undoubtedly instances of recovery when the bleeding has taken place at the stage of maturation. Bleeding from the mucous membranes is also common in this form, and the great majority of the cases prove fatal, usually on the seventh, eighth, or ninth day.

There is a form of hæmorrhagic small-pox in which bleeding takes place into the pocks in the vesicular stage and is followed by a rapid abortion of the rash and a speedy recovery. Six instances of this kind came under my observation.* In four the hæmorrhage took place on the fourth day; in two on the fifth day, just at the time of transition of the papule into the vesicle. Extravasation takes place chiefly into the pocks on the lower extremities and trunk, in only two instances occurring in those of the arms. The eruption in all proved abortive, and no patients under my care with an equal extent of eruption made such rapid recoveries. With these cases are to be grouped those in which the hæmorrhages occur in the pustules of the legs in patients who have in their delirium got out of bed and wandered about. This modified form of hæmorrhagic small-pox is also described by Scheby-Buck.

3. **Varioloid.**—This term is applied to the modified form of small-pox which affects persons who have been vaccinated. It may set in with abruptness and severity, the temperature reaching 103°. More commonly it is in every respect milder in its initial symptoms, though the headache and backache may be very distressing. The papules appear on the evening of the third or on the fourth day. They are few in number and may be confined to the face and hands. The fever drops at once and the patient feels perfectly comfortable. The vesiculation and maturation of the pocks take place rapidly and there is no secondary fever. There is rarely any scarring. As a rule, when small-pox attacks a person who has been vaccinated within five or six years the disease is mild, but there are instances in which it is very severe, and it may even prove fatal.

There are several forms of rash; thus in what has been known as horn-pox, crystalline pox, and wart-pox the papules come out in numbers on the third or fourth day, and by the fifth or sixth day have dried to a hard, horny consistence.

Writers describe a *variola sine eruptione*, which is met with during epidemics in young persons who have been well vaccinated, and who present simply the initial symptoms of fever, headache and backache. In a somewhat extensive experience in Montreal I do not remember to have met with an instance of this kind or to have heard of one.

We do not now see the modified form of small-pox, resulting from inoculation, in which by the seventh or eighth day a pustule forms at the

* Clinical Notes on Small-pox. Montreal, 1876.

seat of inoculation; then general fever sets in, and with it, about the eleventh day, a general eruption, usually limited in degree.

Complications.—Considering the severity of many of the cases and the general character of the disease, associated with multiple foci of suppuration, the complications in small-pox are remarkably few.

Laryngitis is serious in three ways: it may produce a fatal œdema of the glottis; it is liable to extend and involve the cartilages, producing necrosis; and by diminishing the sensibility of the larynx, it allows irritating particles to reach the lower air-passages, where they excite bronchitis or broncho-pneumonia.

Broncho-pneumonia is indeed one of the most common complications, and is almost invariably present in fatal cases. Lobar pneumonia is rare. Pleurisy is common in some epidemics.

The cardiac complications are also rare. In the height of the fever a systolic murmur at the apex is not uncommon; but endocarditis, either simple or malignant, is rarely met with. Pericarditis too is very uncommon. Myocarditis seems to be more frequent, and may be associated with endarteritis of the coronary vessels.

Of complications in the digestive system, parotitis is rare. In severe cases there is extensive pseudo-diphtheritic angina. Vomiting, which is so marked a symptom in the early stage, is rarely persistent. Diarrhœa is not uncommon, as noted by Sydenham, and is very constantly present in children.

Albuminuria is frequent, but true nephritis is rare. Inflammation of the testes and of the ovaries may occur.

Among the most interesting and serious complications are those pertaining to the nervous system. In children convulsions are common. In adults the delirium of the early stage may persist and become violent, and finally subside into a fatal coma. Post-febrile insanity is occasionally met with during convalescence, and very rarely epilepsy. Many of the old writers spoke of paraplegia in connection with the intense backache of the early stage, but it is probably associated with the severe agonising lumbar and crural pains and is not a true paraplegia. It must be separated from the form occurring in convalescence, which may be due to peripheral neuritis or to a diffuse myelitis (Westphal). The neuritis may as in diphtheria involve the pharynx alone, or it may be multiple. Of this nature, in all probability, is the so-called pseudo-tabes, or *ataxie-variolique*. Hemiplegia and aphasia have been met with in a few instances, the result of encephalitis.

Among the most constant and troublesome complications of small-pox are those involving the skin. During convalescence boils are very frequent and may be severe. Acne and ecthyma are also met with. Local gangrene in various parts may occur.

Arthritis may develop, usually in the period of desquamation. It is

probably not a genuine rheumatism. Acute necrosis of the bone is sometimes met with.

Special Senses.—The eye affections which were formerly so common and serious are not now so frequent, owing to the care which is given to keeping the conjunctivæ clean. A catarrhal and purulent conjunctivitis is common in severe cases. The secretions cause adhesions of the eyelids, and unless great care is taken a diffuse keratitis is excited, which may go on to ulceration and perforation. Iritis is not very uncommon. Otitis media is an occasional complication, and usually results from an extension of disease through the Eustachian tubes.

Prognosis.—In unprotected persons small-pox is a very fatal disease. In different epidemics the death-rate is from 25 to 35 per cent. The hæmorrhagic form is invariably fatal, and a majority of those attacked with the severer confluent forms die. In young children it is particularly fatal. In the Montreal epidemic of 1885 and 1886, of 3,164 deaths there were 2,717 under ten years. The intemperate and debilitated succumb more readily to the disease. As Sydenham observed, the danger is directly proportionate to the intensity of the disease on the face and hands. "When the fever increases after the appearance of the pustules, it is a bad sign; but, if it is lessened on their appearance, that is a good sign" (Rhazes). In the confluent cases, when maturation does not proceed and the pocks are flat and if hæmorrhage occurs, the outlook is usually bad. In such cases the general symptoms are apt to be severe. Very high fever, with delirium and subsultus, are symptoms of ill omen. The disease is particularly fatal in pregnant women and abortion usually takes place. It is not, however, uniformly fatal, and I have twice known severe cases to recover after miscarriage. Moreover, abortion is not inevitable. Very severe pharyngitis and laryngitis are fatal complications.

Death results in the early stage from the action of the poison upon the nervous system. In the later stages it usually occurs about the eleventh or twelfth day, at the height of the eruption. In children, and occasionally in adults, the laryngeal and pulmonary complications prove fatal.

Diagnosis.—During an epidemic, the initial chill, followed by fever, headache, vomiting, and the severe pain in the back, are symptoms which should put the attending physician on his guard. Mistakes arise in the initial stage owing to the presence of the scarlatinal or measly rashes which may be extremely deceptive. The scarlatinal rash has not always the intensity of the true rash of this disease. In my Montreal experience I did not meet with an instance in which this rash led to an error, though I heard of several cases in which the mistake was made. These are doubtless the instances to which the older writers refer of scarlet fever and small-pox occurring together. The measly rash cannot always be distinguished from true measles, instances of which may be mistaken for the initial rash. I found in the ward one morning a young man who had been sent in on the previous evening with a diagnosis of small-pox. He

had a fading macular rash with distinct small papules, which had not however the shotty hardness of variola. In the evening this rash was less marked, and as I felt sure that a mistake had been made, he was disinfected and sent home. In another instance a child believed to have small-pox was admitted, but it proved to have simply measles. Neither of these cases took small-pox. In a third case, which I saw at the City Hospital, the mottled papular rash was mistaken for small-pox and the young man sent to the hospital. I saw him the day after admission, when there was no question that the disease was measles and not variola. Less fortunate than the other cases, he took small-pox in a very severe form. The general condition of the patient and the nature of the prodromal symptoms are often better guides than the character of the rash. In any case it is not well, as a rule, to send a patient to a small-pox hospital until the characteristic papules appear about the forehead and on the wrists.

In the most malignant type of hæmorrhagic small-pox the patient may die before the characteristic rash develops, though as a rule small, shotty papules may be felt about the wrists or at the roots of the hair. In only one of twenty-seven cases of hæmorrhagic small-pox, in which death occurred on the third day, did inspection fail to reveal the papules. In three cases in which death took place on the fourth day the characteristic rash was beginning to appear.

The disease may be mistaken for cerebro-spinal fever, in which purpuric symptoms are not uncommon. A four-year-old child was taken suddenly ill with fever, pains in the back and head, and on the second or third day petechiæ appeared on the skin. There was retraction of the head, and marked rigidity of the limbs. The hæmorrhages became more abundant; and finally hæmatemesis occurred and the child died on the sixth day. At the post-mortem there were no lesions of cerebro-spinal fever and in the deeply hæmorrhagic skin the papules could be readily seen. The post-mortem diagnosis of small-pox was unhappily confirmed by the mother taking the disease and dying of it.

It might be thought scarcely possible to mistake any other disease for small-pox in the pustular stage. Yet I had an instance of a young man sent to me with a copious pustular eruption, chiefly on the trunk and covered portions of the body, which, so far as the pustules themselves were concerned, was almost identical with that of variola; but the history and the distribution left no question that it was a pustular syphilide. It is not to be forgotten, however, that fever, which was absent in this case, may be present in certain instances of diffuse pustular syphilis. Lastly, chicken-pox and small-pox may be confounded. Indeed, sometimes it is not easy to distinguish between them, though in well-defined cases of varicella the more vesicular character of the pustules, their irregularity, the short stage of invasion, the slight constitutional disturbance, and the greater intensity of the rash on the trunk, should make the diagnosis clear. It is stated that the Chicago case, which was the starting-point in Montreal of the

epidemic of 1885, was regarded as varicella and not isolated. If so, the mistake was one which led to one of the most fatal of modern outbreaks of the disease.

Glanders in the pustular form has been mistaken for small-pox, and I know of an instance (during an epidemic) which was isolated on the supposition that it was variola.

Treatment.—In the interests of public health cases of small-pox should invariably be removed to special hospitals, since it is impossible to take the proper precautions in private houses. The general hygienic arrangements of the room should be suitable for an infectious disease. All unnecessary furniture and the curtains and carpets should be removed. The greatest care should be taken to keep the patient thoroughly clean, and the linen should be frequently changed. The bedclothing should be light. It is curious that the old-fashioned notion, which Sydenham tried so hard to combat, that small-pox patients should be kept hot and warm, still prevails; and I have frequently had to protest against the patient being, as Sydenham expresses it, stifled in his bed. Special care should be taken to sterilize thoroughly everything that has been in contact with the patient.

In the early stage the pain in the back and limbs requires opium, which, as advised by Sydenham, may be freely given. The diet should consist of milk and broths, and of "all articles which give no trouble to digestion." Cold drinks may be freely given. Barley-water and the Scotch borse (oatmeal and water) are both nutritious and palatable. After the preliminary vomiting, which is often very hard to check by ordinary measures, the appetite is usually good, and, if the throat is not very sore, patients with the confluent form take nourishment well. In the hæmorrhagic cases the vomiting is usually aggravating and persistent.

The fever when high must be kept within limits, and it is best to use either cold sponging or the cold bath. When the pyrexia is combined with delirium and subsultus, the patient should be placed in a bath at 70°, and this repeated as often as every three hours if the temperature rises above 103°. When it is not practicable to give the cold bath, the cold pack can be employed. These measures are much preferable in small-pox to the administration of medicinal antipyretics.

The treatment of the eruption has naturally engaged the special attention of the profession. The question of the preventing of pitting, so much discussed, is really not in the hands of the physician. It depends entirely upon the depth to which the individual pustules reach. After trying all sorts of remedies, such as puncturing the pustules with nitrate of silver, or treating them with iodine and various ointments, I came to Sydenham's conclusion that in guarding the face against being disfigured by the scars "the only effect of oils, liniments, and the like, was to make the white scurfs slower in coming off." There is, I believe, something in protecting

the ripening papules from the light, and the constant application on the face and hands of lint soaked in cold water, to which antiseptics such as carbolic acid or bichloride may be added, is perhaps the most suitable treatment. It is very pleasant to the patient, and for the face it is well to make a mask in lint, which can then be covered with oiled silk. When the crusts begin to form, the chief point is to keep them thoroughly moist, which may be done by oil or glycerin. This prevents the desiccation and diffusion of the flakes of epidermis. Vaseline is particularly useful, and at this stage may be freely used upon the face. It frequently relieves the itching also. For the odor, which is sometimes so characteristic and disagreeable, the dilute carbolic solutions are probably best. If the eruption is abundant on the scalp, the hair should be cut short to prevent matting and decomposition of the crusts. During convalescence frequent bathing is advisable, because it helps to soften the crusts. The care of the eyes is particularly important. The lids should be thoroughly cleansed three or four times a day, and the conjunctivæ washed with some antiseptic solution. In the confluent cases, when the eyelids are much swollen and the lids glued together, it is only by watchfulness that keratitis can be prevented. The mouth and throat should be kept clean, and if crusts form in the nose they should be softened by frequent injections. Ice can be given, and is very grateful when there is much angina. In moderate cases, so soon as the fever subsides the patient should be allowed to get up, a practice which Sydenham warmly urged. The diarrhœa, when severe, should be checked with paregoric. When the pulse becomes feeble and rapid, stimulants may be freely given. The delirium is occasionally maniacal and may require chloroform, but for the nervous symptoms the bath or cold pack is the best. For the severe hæmorrhages of the malignant cases nothing can be done, and it is only cruel to drench the unfortunate patient with iron, ergot, and other drugs. Symptoms of obstruction in the larynx, usually from œdema, may call for tracheotomy. In the late stages of the disease, should the patient be extremely debilitated and the subject of abscesses and bed-sores, he may be placed on a water-bed or treated by the continuous warm bath. During convalescence the patient should bathe daily and use carbolic soap freely in order to get rid of the crusts and scabs. The patient should not be considered free from danger to others until the skin is perfectly smooth and clean, and free from any trace of scabs. I have not mentioned any of the so-called specifics or the internal antiseptics, which have been advised in such numbers; because, so far as I know, the experience of those who have seen the most of the disease does not favor their use.

V. VACCINIA (*Cow-pox*)—VACCINATION.

Definition.—An eruptive disease of the cow, the virus of which, inoculated into man (vaccination), produces a local pock with constitutional disturbance, which affords protection, more or less permanent, from small-pox.

The vaccine is got either directly from the calf—animal lymph—in which the disease is propagated at regular stations, or is obtained from persons vaccinated (humanised lymph).

It was in 1798 that Edward Jenner, a friend and pupil of Hunter, practising in Gloucestershire, announced that persons accidentally inoculated with the cow-pox were subsequently insusceptible to small-pox. From that time the process has extended over the civilized world and proved an incalculable boon to humanity. For many years arm-to-arm vaccination was practised, or the lymph was collected from the vesicle of a child, or the dried scabs were used. The humanised lymph in all probability underwent changes and was certainly more frequently followed by evil results. Of late years animal vaccination has superseded it in great part, and now the lymph is derived either directly from the calf or from one or two removes.

The precise nature of the vaccination virus is as yet unknown. Several forms of micro-organisms have been isolated, and Quist has cultivated micrococci which, he states, produce in the child a typical vaccine vesicle. Several attempts have since been made to isolate the virus, but without definite success. Ernst and Martin, of Boston, have isolated from the bovine lymph a germ which grows on culture media and produces, when inoculated in the calf or in children, characteristic vesicles.

Phenomena of Vaccination.—In a primary vaccination, at the end of twenty-four or thirty-six hours there is seen at the point of insertion of the virus a slight papular elevation surrounded by a reddish zone. The papule gradually increases and on the fifth or sixth day shows a definite vesicle, the margins of which are raised while the centre is depressed. By the eighth day the vesicle has attained its maximum size. It is round and distended with a limpid fluid, the margin hard and prominent, and the umbilication is more distinct. By the tenth day the vesicle is still large and is surrounded by an extensive areola. The skin is also swollen, indurated, and often painful. On the eleventh or twelfth day the hyperæmia diminishes, the lymph becomes more opaque and begins to dry. By the end of the second week the vesicle is converted into a brownish scab which gradually becomes dry and hard, and in about a week (that is, about the twenty-first or twenty-fifth day from the vaccination) separates and leaves a circular pitted scar. If the points of inoculation have been close together, the vesicles fuse and may form a large combined vesicle. Constitutional symptoms of a more or less marked degree follow the vaccination. Usually on the third or fourth day the temperature rises, and may persist, increasing until the eighth or ninth day. In children it is common

to have with the fever restlessness, particularly at night, and irritability; but as a rule these symptoms are trivial. If the inoculation is made on the arm, the axillary glands become large and sore; if on the leg, the inguinal glands. The above may be taken as representing the typical course of vaccination, whether performed with the humanised or with the animal lymph.

Successful vaccination is, for a time at least, an infallible protection against small-pox. The duration of the immunity is extremely variable, differing in different individuals. In some instances it is permanent, but a majority of persons within ten or twelve years again become susceptible.

Revaccination should be performed between the tenth and fifteenth year, and whenever small-pox is epidemic. The susceptibility to revaccination is curiously variable, and when small-pox is prevalent it is not well, if unsuccessful, to be content with a single attempt. The vesicle in revaccination is usually smaller, has less induration and hyperæmia, and the resulting scar is less perfect. Particular care should be taken to watch the vesicle of revaccination, as it not infrequently happens that a spurious pock is formed, which reaches its height early and dries to a scab by the eighth or ninth day. The constitutional symptoms in revaccination are sometimes quite severe.

An irregular course is uncommon in primary vaccination, but we occasionally meet with instances in which the vesicle develops rapidly with much itching, has not the characteristic flattened appearance, the lymph early becomes opaque, and the crust forms by the seventh or eighth day. In such cases the operation should again be performed with fresh lymph.

Complications.—In unhealthy subjects, or as a result of uncleanliness, or sometimes injury, the vesicles inflame and deep excavated ulcers result. Sloughing and deep cellulitis may follow. In debilitated children there may be with this a purpuric rash. Erysipelas may occur, or there may be deep gangrenous ulceration. Such instances are rare, but I have seen two which proved fatal. In one there was deep sloughing and in the other erysipelas. Cases of local dermatitis must not be mistaken for erysipelas. Among the most common complications are certain skin eruptions, some of which are due to the vaccine virus; others result from a mixed infection. Vaccine vesicles not infrequently break out in the immediate vicinity of the primary sores. Less commonly there is a general eruption of vesicles—generalized vaccinia—due to absorption of the virus. More frequent, perhaps, is the erythematous or roseolous rash. Contagious impetigo can also be inoculated with the virus, and may appear as a general eruption.

A question of special importance with reference to vaccination is the transmission of other diseases. For a time physicians were unwilling to acknowledge that constitutional disorders could be transmitted by vaccination, but it is now universally recognized that such transmission may take place, and this has emphasised the scrupulous care which should be taken in the performance of the operation.

Vaccino-Syphilis.—For a knowledge of this most serious of all accidents during vaccination we are largely indebted to Jonathan Hutchinson. It is a true instance of a mixed infection. The vaccine vesicles take as a rule their usual course, and it is not until they have healed or are in process of healing that the local changes characteristic of syphilis are manifested. The fact that syphilis may be transmitted in this way should put the practitioner on his guard in selecting humanised lymph. He should take it only from subjects with whose constitution he is perfectly familiar. Fortunately, the instances are extremely rare. They are, in fact, much less frequent than is usually supposed, and in a majority of the cases in which vaccino-syphilis is suspected the condition is really that of inflamed and indurated vaccinal ulcer. As the subject is of daily interest to the practitioner, and one which he may at any moment be called upon to decide, I here insert a table of differential features between vaccinal ulcers and vaccino-syphilis, and between the vaccination rashes and the secondary syphilitic eruptions, compiled by C. E. Shelly * from Fournier's lectures.

VACCINO-SYPHILIS.	VACCINATION ULCERS.
Chancre developed on the site of usually one or two only of the vaccination punctures.	Ulceration affects all the punctures as a rule.
Inflammation is slight.	Inflammation and ulceration severe.
Loss of substance superficial only.	Ulcer deeply excavated.
Suppuration scanty or absent, scabs or crusts formed.	Much suppuration.
Border of chancre smooth, slightly elevated, gradually merging into floor.	Margin of ulcer irregular, as in "soft chancre."
Surface of floor smooth.	Floor of ulcer uneven, suppurating.
Induration "parchment-like" and specific, not merely inflammatory.	Induration inflammatory only.
Inflammatory areola very slight.	Areola inflammatory and erysipelatous in character.
Gland swelling constant, indolent (syphilitic) bubo.	Gland swelling often absent; if present, merely inflammatory.
Complications rare.	Complications—sloughing, erysipelas, etc.—often present.
Chancre never developed before the fifteenth day after vaccination; usually not until after three to five weeks; still in its earlier stage twenty days after vaccination.	Ulceration is present twelve or fifteen days after vaccination and is fully developed by the twentieth day after vaccination.

* Fowler's Dictionary of Medicine. Article Vaccination.

SECONDARY SYPHILITIC ERUPTION due to true vaccino-syphilis.	VACCINATION RASHES (including roseola vaccinalis, miliaria vaccinalis, vaccinia bullosa, vaccinia hæmorrhagica); also accidental eruptions — rubeola, scarlatina, lichen, urticaria, etc.
Appears, at the earliest, nine or ten weeks after vaccination.	A true vaccinal rash appears between the ninth and fifteenth day after vaccination.
Requires, in every case, the pre-existence of a specific ulcer (chancre) at the site of vaccination.	Absence of inoculation chancre.
Exhibits the characters of a true specific eruption.	Eruption does not exhibit specific characters.
Fever often slight.	Fever always present.
Lasts for a long time.	Evanescent.
Usually accompanied by specific appearances on mucous membranes.	

VACCINO-SYPHILIS.	HEREDITARY SYPHILIS SHOWING ITSELF ABOUT THE TIME OF VACCINATION.
Begins with a local infection, chancre and indolent bubo.	No chancre; begins with general phenomena.
Typical development in four stages, viz., incubation, chancre, second incubation, generalization (secondary eruptions, etc.).	Has no typical development in connection with vaccination.
Never appears earlier than the ninth or tenth week after vaccination.	Time of development quite independent of vaccination.
	Is attended by the characteristic syphilitic bodily aspect.
	Other manifestations of hereditary syphilis may be present.
	The history may indicate syphilis.

Choice of Lymph.—Humanised lymph should be taken on the eighth day and only from perfectly formed unbroken vesicles, which have had a typical course, and have not yet developed areolæ. Pricking or scratching the surface, the greatest care being taken not to draw blood, allows the lymph to exude, and it may then be collected on ivory points or in capillary tubes. The child from which the lymph is taken should be healthy, strong, and known to be of good stock, free from tuberculous or syphilitic taint. Under these circumstances humanised lymph, one or

two removes from the calf, is usually very satisfactory in its action and is perfectly reliable.

In the case of the calf the most scrupulous care should be exercised in the vaccine farms to secure animals which are healthy and strong. The risk, however, that the calf has any disease which can be transmitted to man is exceedingly slight, as tuberculosis is very rare in cattle when young. Unquestionably, however, there may be risk in the case of a calf born of tuberculous parents, and special care should be taken in the selection of proper animals. There is no essential difference in the pocks which follow humanised lymph and bovine lymph. It was, I believe, a common experience in Montreal that children inoculated with bovine lymph had more constitutional disturbance and often sorer arms than those vaccinated with humanised lymph at one or two removes.

In the performance of the operation that part of the arm about the insertion of the deltoid is usually selected. Mothers "in society" prefer to have girl babies vaccinated on the leg. The skin should be cleansed and put upon the stretch. Then, with a lancet or the ivory point, cross-scratches should be made in one or more places. When the lymph has dried on the points it is best to moisten it in warm water. The clothing of the child should not be adjusted until the spot has dried, and it should be protected for a day or two with lint or a soft handkerchief. If erysipelas is prevalent, or if there are cases of suppuration in the same house, it is well to apply a pad of antiseptic cotton. Vaccination is usually performed at the second or third month. If unsuccessful, it should be repeated from time to time. A person exposed to the contagion of small-pox should always be revaccinated. This, if successful, will usually protect; but not always, as there are many instances in which, though the vaccination takes, variola also appears.

The Value of Vaccination.—Vaccination is not claimed to be an invariable and permanent preventive of small-pox, but in an immense majority of cases successful inoculation renders the person for many years insusceptible. Communities in which vaccination and revaccination are thoroughly and systematically carried out are those in which small-pox has the fewest victims. On the other hand communities in which vaccination and revaccination are persistently neglected are those in which epidemics are most prevalent. In the German army the practice of revaccination has stamped out the disease. Nothing in recent times has been more instructive in this connection than the fatal statistics of Montreal. The epidemic which started in 1870–'71 was severe in Lower Canada, and persisted in Montreal until 1875. A great deal of feeling had been aroused among the French Canadians by the occurrence of several serious cases of ulceration, possibly of syphilitic disease, following vaccination; and several agitators, among them a French physician of some standing, aroused a popular and wide-spread prejudice against the practice. There were indeed vaccination riots. The introduction of animal lymph was distinctly

beneficial in extending the practice among the lower classes, but compulsory vaccination could not be carried out. Between the years 1876 and 1884 a considerable unprotected population grew up and the materials were ripe for an extensive epidemic. The soil had been prepared with the greatest care and it only needed the introduction of the seed, which in due time came as already stated with the Pullman-car conductor from Chicago, on the 28th of February, 1885. Within the next ten months thousands of persons were struck with the disease, and 3,164 died.

Although the effects of a single vaccination may wear out, as we say, and the individual again become susceptible to small-pox, yet the mortality in such cases is very much lower than in persons who have never been vaccinated. The mortality in persons who have been vaccinated is from 6 to 8 per cent, whereas in the unvaccinated it is at least 35 per cent. Marson pointed out some years ago that there is a definite ratio between the number of deaths and the number of good vaccination marks in post-vaccinal small-pox. With good marks the mortality is between 3 and 4 per cent, and with indifferent marks at least 10 or 11 per cent.

VI. VARICELLA (*Chicken-pox*).

Definition.—An acute contagious disease of children, characterised by an eruption of vesicles on the skin.

Etiology.—The disease occurs in epidemics, but sporadic cases are also met with. It may prevail at the same time as small-pox or may follow or precede epidemics of this disease. An attack of chicken-pox is no protection against small-pox. It is a disease of childhood; a majority of the cases occur between the second and sixth years. It is rarely seen in adults. The bacteriological examination of the vesicles has shown the presence of micrococci in the contents of the vesicles, but the specific germ has not yet been discovered.

There can be no question that varicella is an affection quite distinct from variola and without at present any relation whatever to it. An attack of the one does not confer immunity from an attack of the other. The case which Sharkey reported is of special importance in this connection. A boy, aged five, was admitted to St. Thomas' Hospital with a vesicular eruption, and was isolated in a ward on the same floor as the small-pox ward. The disease was pronounced chicken-pox, however, by Sir Risdon Bennett and Dr. Bristowe. The patient was then removed and vaccinated, with a result of four vesicles which ran a pretty normal course. On the eighth day from the vaccination the child became feverish. On the following day the papules appeared and the child had a well-developed attack of small-pox with secondary fever.

Symptoms.—After a period of incubation of ten or fifteen days the child becomes feverish and in some instances has a slight chill. There

may be vomiting and pains in the back and legs. Convulsions are rare. The eruption usually develops within twenty-four hours. It is first seen upon the trunk, either on the back or on the chest. I have seen it, however, appear first on the forehead and face. At first in the form of raised red papules, they are in a few hours transformed into hemispherical vesicles containing a clear or turbid fluid. There is no umbilication as in the vesicles of small-pox. They are often ovoid in shape and look more superficial than the variolous vesicles. The skin in the neighborhood is neither infiltrated nor hyperæmic. At the end of thirty-six or forty-eight hours the contents of the vesicles are purulent. They begin to shrivel and during the third and fourth days are converted into dark brownish crusts, which fall off and as a rule leave no scar. Fresh crops appear during the first two or three days of the illness, so that on the fourth day one can usually see pocks in all stages of development and decay. They are always discrete and the number may vary from eight or ten to several hundreds. As in variola, a scarlatinal rash occasionally precedes the development of the eruption.

There are one or two modifications of the rash which are interesting. The vesicles may become very large and develop into regular bullæ, looking not unlike ecthyma. The irritation of the rash may be excessive, and if the child scratches the pocks ulcerating sores may form, which on healing leave ugly scars. Indeed, cicatrices after chicken-pox are not so very uncommon. They are in my experience more common than after varioloid. The fever in varicella is slight, but it does not as a rule disappear with the appearance of the rash. The course of the disease is in a large majority of the cases favorable and no ill effects follow. The disease may recur in the same individual. There are instances in which a person has had three attacks.

There are one or two interesting complications of chicken-pox. In delicate children, particularly the tuberculous, gangrene may occur about the vesicles (Abercrombie).

Cases have been described (Andrew) of hæmorrhagic varicella with cutaneous ecchymoses and bleeding from the mucous membranes.

Nephritis may occur. Infantile hemiplegia has developed during an attack of the disease.

The *diagnosis* is as a rule easy, particularly if the patient has been seen from the outset. When a case comes under observation for the first time with the rash well out, there may be considerable difficulty. The pocks in varicella are more superficial, more bleb-like, have not the infiltrated areola about them, and may usually be seen in all stages of development. They rarely at the outset have the hard, shotty feeling of small-pox. The general symptoms, the greater intensity of the onset, the prolonged period of invasion, and the more frequent occurrence of prodromal rashes in small-pox are important points in the diagnosis.

No special *treatment* is required. If the rash is abundant on the

face great care should be taken to prevent the child from scratching the pustules. A soothing lotion should be applied on lint.

VII. SCARLET FEVER.

Definition.—An infectious disease characterised by a diffuse exanthem and an angina of variable intensity.

Etiology.—We owe the recognition of scarlet fever as a distinct disease to Sydenham, before whose time it was confounded with measles. It is a wide-spread affection, occurring in nearly all parts of the globe and attacking all races.

The disease occurs sporadically from time to time, and then under unknown conditions becomes wide-spread. Epidemics vary in severity.

Among predisposing factors age is most important. A large proportion of the cases occur before the tenth year. Of an enormous number of fatal cases tabulated by Murchison over 90 per cent occurred in children under this age. Adults, however, are by no means exempt. Very young infants are rarely attacked. A certain number exposed to the contagion escape. In a family of children all more or less exposed one or two may not take the disease, whereas all as a rule, if exposed, take the measles. The susceptibility seems to vary in families, and we meet occasionally with sad instances in which three or more members of a family succumb in rapid succession.

Males and females are equally affected.

Epidemics prevail at all seasons, but perhaps with greater intensity in autumn and winter.

The contagion of scarlet fever is probably not developed until the eruption appears, and is particularly to be dreaded during desquamation. No doubt the poison is spread largely by the fine scaly particles which are diffused with the dust throughout the room. Even late in the disease, after desquamation has been apparently completed, a patient has conveyed the contagion. The poison clings with great persistence to clothing of all kinds and to articles of furniture in the room. In no disease is a greater tenacity displayed. Bedding and clothes which have been put away for months or even for years may, unless thoroughly disinfected, convey contagion. Physicians, nurses, and others in contact with the sick may carry the poison to persons at a distance. It is remarkable that in the case of physicians this does not more frequently occur. I know of but one instance in which I carried the contagion of this disease. The poison probably is not widely spread in the atmosphere. Observations have been recently made which indicate that the poison may be conveyed in milk. The epidemic investigated by Power and Klein in London in 1885 was traced by them to milk obtained from a dairy at Hendon, in which the cows were found to be suffering from a vesicular affection of

the udder. The nature of this disease of the cow is doubtful, however. Crookshank maintains that it was cow-pox, and had nothing to do with scarlet fever.

Some writers maintain that scarlet fever may be associated with defective house-drainage. Possibly the virus may occasionally gain entrance in this way.

The attack does not necessarily protect permanently. There are instances of a second and even a third attack.

Surgical and puerperal scarlatinas, so called, demand a word under this section. While scarlet fever may attack a person after operation, or a woman in childbed, the majority of the cases described as such represent, I believe, only the red rash of septicæmia. In the cases which I have seen the rash was rarely so widespread as in scarlet fever; the tongue had not the special features, nor was the throat affected. Desquamation is no criterion, as it occurs whenever hyperæmia of the skin persists for any length of time. It is interesting to note that these cases have become rare with the gradual disappearance of septicæmia. I. E. Atkinson suggests that these rashes are in many cases due to quinine.

Attempts to determine the specific germ of scarlet fever have so far proved ineffectual. Occasionally streptococci are found in the blood, and in fatal cases they are found in the lymph-glands and in the kidneys. It will no doubt soon be determined whether Loeffler's bacillus of diphtheria exists in the pseudo-membranes in the throat. Cornil and Babes state that it does, and that in the angina without diphtheria there are only streptococci. In some cases the bacillus of diphtheria has been found late in the disease. The point is one of great importance, and could be settled by careful observations.

Morbid Anatomy.—Except in the hæmorrhagic form, the skin after death shows no traces of the rash. There are no specific lesions. Those which occur in the internal organs are due partly to the fever and partly to infection with pus-organisms.

The anatomical changes in the throat are those of simple inflammation, follicular tonsillitis, and, in extreme grades, of pseudo-membranous angina. In severe cases there is intense lymphadenitis and much inflammatory œdema of the tissues of the neck, which may go on to suppuration, or even to gangrene. Streptococci are found abundantly in the glands and in the areas of suppuration. Of changes in the digestive organs, a catarrhal state of the gastro-intestinal mucosa is not uncommon. The liver may show interstitial changes (Klein). The spleen is often enlarged.

Endocarditis and pericarditis are not infrequent. Myocardial changes are less common. The renal changes are the most important, and have been thoroughly studied by Coats, Klebs, Wagner, and others. The special nephritis of the disease will be considered with the diseases of the kidney.

Affections of the respiratory organs are not frequent. When death

results from the pseudo-membranous angina, broncho-pneumonia is not uncommon. Cerebro-spinal changes are rare.

Symptoms.—Incubation.—On this point there is great discrepancy. The period is undoubtedly very variable. From three to twelve days is probably the limit, though it may in exceptional cases be extended. In one case, the circumstances of which made it perfectly clear that I had myself conveyed the infection, the incubation was twelve days.

Invasion.—The onset is as a rule sudden. It may be preceded by a slight scarcely noticeable indisposition. An actual chill is rare. Vomiting and, in young children, convulsions are common. The fever is intense; rising rapidly, it may on the first day reach 104° or even 105°. The skin is unusually dry and to the touch gives a sensation of very pungent heat. The tongue is furred, and as early as the first day there may be complaint of dryness of the throat. Cough and catarrhal symptoms are uncommon. The face is often flushed and the patient has all the objective features of an acute fever.

Eruption.—Usually on the second day, in some instances within twenty-four hours, the rash develops in the form of scattered red points on a deep subcuticular flush. It appears first on the neck and chest, and spreads so rapidly that by the evening of the second day it may have invaded the entire skin. In pronounced cases the rash at its height has a vivid scarlet hue, quite distinctive and unlike that seen in any other eruptive disease. It is entirely hyperæmic, and the anæmia produced by pressure instantly disappears. In some cases the rash does not become uniform but remains patchy, and intervals of normal skin separate large hyperæmic areas. Tiny papular elevations may sometimes be seen, but they are not so common as in measles. At the height of the eruption sudaminal vesicles may develop, the fluid of which may become turbid. The entire skin may at the same time be covered with small yellow vesicles on a deep red background. Pronounced cases of this type were called by the older writers *scarlatina miliaris*.

Occasionally there are petechiæ, which in the malignant type of the disease become wide-spread and large. The eruption does not always appear upon the face. There may be a good deal of swelling of the skin which feels uncomfortable and tense. The itching is variable; not as a rule intense at the height of the eruption. After persisting for two or three days the rash gradually fades. The rash can often be seen on the mucous membranes of the palate, the cheeks and the tonsils, giving to these parts a vivid red, punctiform appearance. The tongue is red at the tip and edges, furred in the centre; and through the white fur are often seen the swollen papillæ, which give the so-called "strawberry" appearance to the tongue. The breath often has a very heavy, sweet odor.

The pharyngeal symptoms vary extremely. There may be—

1. Slight redness, with swelling of the pillars of the fauces and of the tonsils.

2. A more intense grade of swelling and infiltration of these parts with a follicular tonsillitis.

3. Membranous angina with intense inflammation of all the pharyngeal structures and swelling of the glands below the jaw, and in very severe cases a thick brawny induration of all the tissues of the neck.

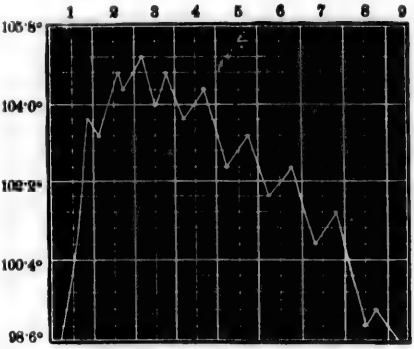


CHART IX.—Scarlet fever (Strümpell).

The fever, which sets in with such suddenness and intensity, may reach 105° or even 106°. It persists with slight morning remissions, gradually declining with the disappearance of the rash. In mild cases the temperature may not reach 103°; on the other hand, in very severe cases there may be hyperpyrexia, the thermometer registering 108° or even before death 109°.

The pulse presents the ordinary febrile characters, ranging in children from 120° to 150°, or even higher. The respirations show an increase proportionate to the intensity of the fever. The gastro-intestinal symptoms are not marked after the initial vomiting, and food is usually well taken. In some instances there are abdominal pains. The edge of the spleen may be palpable. The liver is not often enlarged. With the initial fever nervous symptoms are present in a majority of the cases; but as the rash comes out the headache and the slight nocturnal wandering disappear. The urine has the ordinary febrile characters, being scanty and high colored. Albuminuria is by no means infrequent during the stage of eruption, but the amount is slight. Careful examination of the urine should be made every day. There is no cause for alarm in the slight trace of albumen which is so often present, not even if it is associated with a few tube-casts.

Desquamation.—With the disappearance of the rash and the fever the skin looks somewhat stained, is dry, a little rough, and gradually the upper layer of the cuticle begins to separate. The process usually begins about the neck and chest, and flakes are gradually detached. The degree and character of the desquamation bear some relation to the intensity of the eruption. When the latter has been very vivid and of long-standing, large flakes may be detached. In rare instances the hair and even the nails have been shed. It must not be forgotten that there are cases in which the desquamation has been prolonged, according to Trousseau even to the seventh or eighth week. The entire process lasts from ten to fifteen or even twenty days.

There are cases of exceptional mildness in which the rash may be

scarcely perceptible. During epidemics, when several children of a household are affected, it sometimes happens that a child sickens as if of scarlet fever, and has a sore throat and the "strawberry tongue" without the development of any rash. This is the so-called *scarlatina sine eruptione*.

These slight cases of scarlet fever may be followed by the severest attacks of nephritis.

MALIGNANT SCARLET FEVER.

Ataxic Form.—This presents all the characteristics of an acute intoxication. The patient overwhelmed by the intensity of the poison may die within twenty-four or thirty-six hours. The disease sets in with great severity—high fever, extreme restlessness, headache, and delirium. The temperature may rise to 107° or even 108°, and rare cases have been observed in which the thermometer has registered even higher. Convulsions may occur in children. The initial delirium rapidly gives place to coma. The dyspnœa may be urgent; the pulse is very rapid and feeble.

Hæmorrhagic Form.—In some instances hæmorrhages occur into the skin. There is hæmaturia, and epistaxis. In the erythematous rash there are at first scattered petechiæ, which gradually become more extensive, and ultimately the skin may be universally involved. Death may take place on the second or on the third day. While this form is perhaps more common in enfeebled children, I have twice known it to attack persons apparently in full health.

Anginose Form.—The throat symptoms may appear early and progress rapidly. The fauces and tonsils are swollen. Membranous exudation forms. It may extend to the posterior wall of the pharynx, forward into the mouth, and upward into the nostrils. The glands of the neck rapidly enlarge. Necrosis occurs in the tissues of the throat, the fœtor is extreme, the constitutional disturbance profound, and the child dies with the clinical picture of a malignant diphtheria. Occasionally the membrane extends into the trachea and the bronchi. The Eustachian tubes and the middle ear are usually involved. In cases in which death does not take place rapidly from toxæmia there may be extensive abscess formation in the tissues of the neck and sloughing. In the separation of deep sloughs about the tonsils the carotid artery may be opened, causing fatal hæmorrhage.

As already mentioned, scarlatinal angina, though resembling diphtheria and not to be distinguished from it anatomically, is probably due to the scarlatinal and not to the diphtheritic poison.

Complications and Sequelæ.—(*a*) *Nephritis.*—At the height of the fever there is often a slight trace of albumen in the urine, which is not of special significance. In a majority of cases the kidneys escape without greater damage than occurs in other acute febrile affections.

Nephritis is most common in the second or third week and may develop after a very mild attack. It may be delayed until the third or

fourth week. As a rule, the earlier it develops in the disease the more intense it is. It varies greatly in intensity, and three grades of cases may be recognized:

1. Very severe cases with suppression of urine or the passage of a small quantity of dark bloody urine laden with albumen and tube-casts. Vomiting is constant, there are convulsions, and the child dies with the symptoms of acute uræmia.

2. Less severe cases without any serious acute symptoms. There is a puffy appearance of the eyelids, with slight œdema of the feet; the urine is diminished in quantity, smoky in appearance, and contains albumen and tube-casts. The kidney symptoms then dominate the entire case, the dropsy persists, and there may be effusion into the serous sacs. The case may drag on and become chronic, or the patient may succumb to uræmic accidents. Fortunately, in a majority of the cases the disease yields to judicious treatment and recovery takes place.

3. Cases so mild that they can scarcely be termed nephritis. The urine shows a moderate amount of albumen. There may be tube-casts, rarely blood. The œdema is extremely slight or transient, and the convalescence is scarcely interrupted. Occasionally, however, in these mild attacks serious symptoms may supervene. Œdema of the glottis may prove rapidly fatal, and in one case of the kind a child under my care died of acute effusion into the pleural sacs.

There are instances of œdema without albuminuria or signs of nephritis. Possibly in some of these cases the œdema may be hæmic and due to the anæmia; but there are instances in which marked changes have been found in the kidney after death, even when the urine did not show the features characteristic of nephritis.

(*b*) *Arthritis.*—During the subsidence of the fever, rarely at its height, pains and swelling in the joints may develop and present all the characteristics of acute rheumatism. In all probability it is not however true rheumatism, but is analogous to gonorrhœal synovitis. It may pass on to suppuration, in which case it most commonly involves only a single joint.

(*c*) *Cardiac Complications.*—Simple endocarditis is not uncommon, and many cases of chronic valvular disease originate probably in the latent endocarditis of this disease. Malignant endocarditis is rare. Pericarditis is probably not more frequent, but is less likely to be overlooked than endocarditis. It usually develops during convalescence, and may be sero-fibrinous or purulent. The cardiac complications are sometimes found in association with arthritis. Myocarditis is not uncommon.

(*d*) *Pleurisy* may follow *pneumonia*, though this is rare. More often it occurs during convalescence, is insidious in its course, and as a rule purulent. This serious complication of scarlet fever is not sufficiently recognized. It was one upon which my teacher, R. P. Howard,* in Mont-

* Canada Medical and Surgical Journal, December, 1873.

real, specially insisted in his lectures. Sheriff, in a number of the same journal, reports two cases, occurring at the same time in brothers, one of whom died suddenly after a slight exertion.

(*e*) *Ear Complications.*—These are common and serious. They are due to extension of the inflammation from the throat through the Eustachian tubes. It is one of the most frequent causes of deafness. The severe forms of membranous angina are almost always associated with inflammation of the middle ear, which goes on to suppuration and perforation of the drum. The suppuration may extend to the labyrinth and rapidly produce deafness. In other instances there is suppuration in the mastoid cells. In the necrosis which follows the middle-ear disease, the facial nerve may be involved and paralysis follow. Later, still more serious complications may follow the otitis; such as thrombosis of the lateral sinus, meningitis, or abscess of the brain.

(*f*) *Adenitis.*—In comparatively mild cases of scarlet fever the submaxillary lymph-glands may be swollen. In severer cases the swelling of the neck becomes extreme and extends beyond the limits of the glands. Acute phlegmonous inflammations may occur, leading to wide-spread destruction of tissue, in which vessels may be eroded and fatal hæmorrhage ensue. The suppurative processes may also involve the retro-pharyngeal tissues.

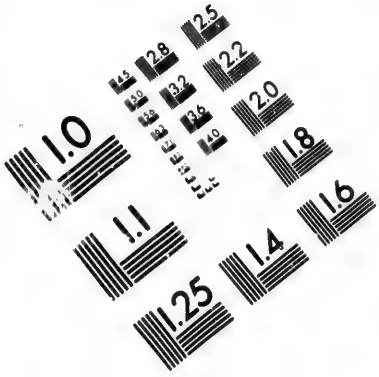
The swelling of the lymph-glands usually subsides, and within a few weeks even the most extensive enlargement gradually disappears. There are rare instances, however, in which the lymphadenitis becomes chronic and the neck remains with a glandular collar which almost obliterates its outline. This may prove intractable to all ordinary measures of treatment. A case came under my observation in which, two years after scarlet fever, the neck was enormously enlarged and surrounded by a mass of firm brawny glands.

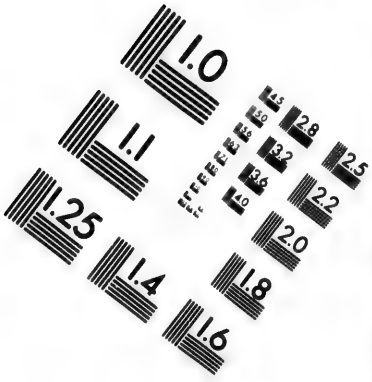
(*g*) *Nervous Complications.*—Chorea occasionally develops in connection with the arthritis and endocarditis. Sudden convulsions followed by hemiplegia may occur. Two instances of progressive paralysis of the limbs with wasting came under my observation at the Philadelphia Infirmary for Nervous Diseases. The history was that of subacute ascending spinal paralysis, but it is probable that they were instances of multiple neuritis. Mental symptoms, mania and melancholia, have been described.

(*h*) Other rare complications and sequelæ are eye affections, symmetrical gangrene, enteritis, and noma.

Diagnosis.—The diagnosis of scarlet fever is not difficult, but there are cases in which the true nature of the disease is for a time doubtful. The following are the most common conditions with which it may be confounded.

1. *Acute Exfoliating Dermatitis.*—This pseudo-exanthem simulates scarlet fever very closely. It has a sudden onset, with fever. The eruption spreads rapidly, is uniform, and after persisting for five or six days





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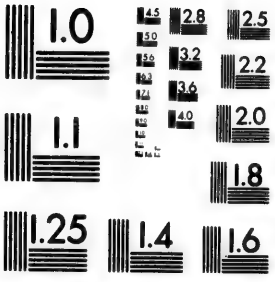

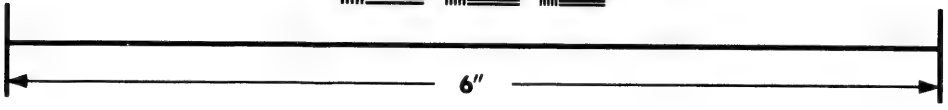

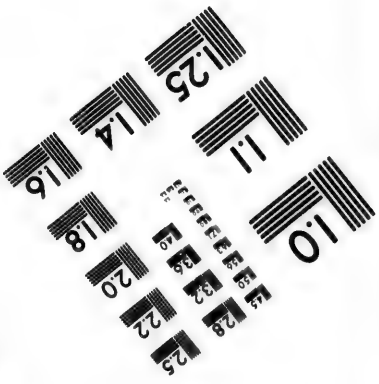

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begins to fade. Even before it has entirely gone, desquamation usually begins. Some of these cases cannot be distinguished from scarlet fever in the stage of eruption. The throat symptoms, however, are usually absent, and the tongue rarely shows the changes which are so marked in scarlet fever. In the desquamation of this affection the hair and nails are commonly affected. It is, too, a disease liable to recur. Some of the instances of second and third attacks of scarlet fever have been cases of this form of dermatitis.

2. *Measles*, which is distinguished by the longer period of invasion, the characteristic nature of the prodromes, and the later appearance of the rash. The greater intensity of the measly rash upon the face, the more papular character, the irregular crescentic distribution, are distinguishing features in a majority of the cases. Other points are the absence of the sore throat in measles and the peculiar character of the desquamation.

3. *Rötheln.*—The rash of rubella is sometimes strikingly like that of scarlet fever, but in the great majority of cases the mistake could not arise. In cases of doubt the general symptoms are our best guide.

4. *Septicæmia.*—As already mentioned, the so-called puerperal or surgical scarlatina shows an eruption which may be identical in appearance with that of true scarlet fever.

5. *Diphtheria.*—The practitioner may be in doubt whether he is dealing with a case of scarlet fever with intense membranous angina, or a true diphtheria with an erythematous rash. The erythema in diphtheria may appear early, before the throat symptoms are well developed, or as they are appearing, in which case it is usually slight and disappears quickly. There is also, when the disease is at its height, a later erythema, which may be very diffuse and intense. The subsequent desquamation can not always be relied upon to make clear the diagnosis, for any intense erythema of sufficient duration will be followed by this process. None of the preceding conditions offer difficulties so great as these cases of angina with erythematous rash, and it may be impossible to determine satisfactorily the true nature of the trouble. Fortunately, so far as treatment is concerned, this does not make much difference. A bacteriological examination of the exudate should be made in doubtful cases.

6. *Drug Rashes.*—These are partial, and seldom more than a transient hyperæmia of the skin. Occasionally they are diffuse and intense, and in such cases very deceptive. They are not associated, however, with the characteristic symptoms of invasion. There is no fever, and with care the distinction can usually be made. They are most apt to follow the use of belladonna, quinine, and iodide of potassium.

Prognosis.—Epidemics differ in severity and the death-rate is extremely variable. Among the better classes the death-rate is much less than in hospital practice. There are physicians who have treated consecutively a hundred or more cases without a death. On the other hand, in hospitals and among the poorer classes the death-rate is considerable,

ranging from 5 to 10 per cent in mild epidemics to 20 or 30 per cent in the very severe.

The younger the child the greater the danger. In infants under one year the death-rate is very high. The great proportion of fatal cases occurs in children under six years of age.

The unfavorable symptoms are very high fever, early mental disturbance with great jactitation, the occurrence of hæmorrhages (cutaneous or visceral), intense pseudo-membranous angina with cervical bubo, and signs of laryngeal obstruction.

Nephritis is always a serious complication and when setting in with suppression of the urine may quickly prove fatal. It is noteworthy, however, that a large majority of the cases of scarlatinal nephritis recover.

Treatment.—The disease cannot be cut short. In the presence of the severer forms we are still too often helpless. There is no disease in which the successful issue and the avoidance of complications depends more upon the skilled judgment of the physician and the care with which his instructions are carried out.

The child should be isolated and placed in charge of a competent nurse. The temperature of the room should be constant and the ventilation thorough. The child should wear a light flannel night-gown, and the bedclothing should not be too heavy. The diet should consist of milk, broths, and fresh fruits, and water should be freely given. With the fall of the temperature, the diet may be increased and the child may gradually return to ordinary fare. When desquamation begins the child should be thoroughly rubbed every day, or every second day, with sweet oil, which prevents the drying and the diffusion of the scales. An occasional warm bath may then be given. At any time during the attack the skin may be sponged with warm water. The patient may be allowed to get up after the temperature has been normal for ten days, but for at least three weeks from this time great care should be exercised to prevent exposure to cold. It must not be forgotten, also, that the renal complications are very apt to develop during the convalescence, and after all danger is apparently past. Ordinary cases do not require any medicine, or at the most a simple fever mixture, and during convalescence a bitter tonic. The bowels should be carefully regulated, either with small doses of calomel or with mild aperients.

Special symptoms in the severe cases call for treatment.

When the temperature is above 103° the extremities may be sponged with tepid water. In severe cases, with the temperature rapidly rising, this will not suffice, and more thorough measures of hydrotherapy should be practised. With pronounced delirium and nervous symptoms the cold-pack should be used. When the temperature is rising rapidly but the child is not delirious, he should be placed in a warm bath, the temperature of which can be gradually lowered. The bath at a temperature of 80° is beneficial. In giving the cold-pack a rubber sheet and a thick layer of

blanket should be laid upon a sofa or a bed, and upon this a sheet, wrung out of cold water. The naked child is then laid upon it and wrapped in the blankets. An intense glow of heat quickly follows the preliminary chilling, and from time to time the blankets may be unfolded and the child sprinkled with cold water. The good effects which follow this plan of treatment are often striking, particularly in allaying the delirium and jactitation, and procuring quiet and refreshing sleep. Parents will object less, as a rule, to the warm bath gradually cooled than to any other form of hydrotherapy. The child may be removed from the warm bath, placed upon a sheet wrung out of tolerably cold water, and then folded in blankets. The ice-cap is very useful and may be kept constantly applied in cases in which there is high fever. Medicinal antipyretics are not of much service in comparison with cold water.

The throat symptoms, if mild, do not require much treatment. Applications may be made with a spray, and if the laryngitis becomes severe the measures should be used which will be mentioned under croup. Cold applications to the neck are to be preferred to hot, though it is sometimes difficult to get a child to submit to them. In connection with the throat symptoms the ears should be specially looked after, and a careful disinfection of the throat by suitable antiseptic solutions should be practised. When the inflammation extends through the tubes to the middle ear, the practitioner should either himself daily examine the conditions of the drum, or, when available, a specialist should be called in to assist him in the case. The careful watching of this membrane day by day and the puncturing of it if the tension becomes too great may save the hearing of the child. With the aid of cocaine the drum is readily punctured. The operation may be repeated at intervals if the pain and distention return. No complication of the disease is more serious than this extension of the inflammatory process to the ear.

The nephritis should be dealt with as in ordinary cases, and indications for treatment will be found under the appropriate section. It is worth mentioning, however, that Jaccoud insists upon the great value of milk diet in scarlet fever as a preventive of nephritis.

Among other indications for treatment in the disease is cardiac weakness, which is usually the result of the direct action of the poison, and is best met by stimulants.

Many specifics have been vaunted in scarlet fever, but they are all useless. J. C. Wilson recommends chloral in one or two grain doses for a child of two or three years.

VIII. MEASLES.

Definition.—An acute, highly infectious disorder, characterised by an initial coryza and a rapidly spreading eruption.

Etiology.—The infection of measles is very intense and immunity against attack not nearly so common as in scarlet fever. It is a disease of childhood, but unprotected adults are liable to the infection. Indeed, measles is more frequent in adults than is scarlet fever. Within the first six months of life the liability is not so marked, though I have known infants of a month and of six weeks to be attacked. The sexes are equally affected. The contagion is communicated by the breath and by the secretions, particularly those of the nose. It may be conveyed by a third person and by fomites.

The disease is practically endemic in large centres of population, and from time to time spreads and prevails epidemically. It occurs at all seasons, but prevails more extensively during the colder months. There is no infectious disease in which recurrence is more frequent. There may be a second, third, or even a fourth attack.

The contagion of the disease is unknown. No one of the various organisms which have been described meets the requirements of Koch's law.

Morbid Anatomy.—Measles itself rarely kills, but the complications and sequelæ combine to make it a very fatal affection in children. There are no characteristic post-mortem appearances. The skin changes are those associated with an intense hyperæmia.

There is a catarrhal condition of the mucous membranes, particularly of the bronchi. The fatal cases show almost invariably either broncho-pneumonia, capillary bronchitis with patches of collapse, or less frequently lobar pneumonia. The bronchial glands are invariably swollen. Pleurisy is less common. During convalescence from measles there is a special liability to tuberculous invasion, and tuberculous broncho-pneumonia claims a large number of victims. The bronchial glands may also be affected.

The gastro-intestinal mucosa may be hyperæmic. Swelling of Peyer's glands is not at all uncommon and may reach a very intense grade in the patches.

Symptoms.—Incubation.—This is about ten days, but the limits are variable, and it may be as long as twenty days. The disease has been frequently inoculated. In such cases the incubation period is less than ten days.

Invasion.—The disease usually begins with symptoms of a feverish cold. There are shiverings (not often a definite chill), marked coryza, sneezing, running at the nose, redness of the eyes and lids, with photophobia, and within twenty-four hours cough. These early catarrhal symptoms are more marked in measles than in any other infectious disease of children. There may be the symptoms so commonly associated with an on-coming fever—nausea, vomiting, and headache. The tongue is

furred. Examination of the throat may show a reddish hyperæmia or in some instances a distinct punctiform rash. Occasionally this spreads over the whole mucous membrane of the mouth with the exception of the tongue. The temperature at this stage is usually high, reaching from 103° to 104°, ascending gradually through the second and third days.

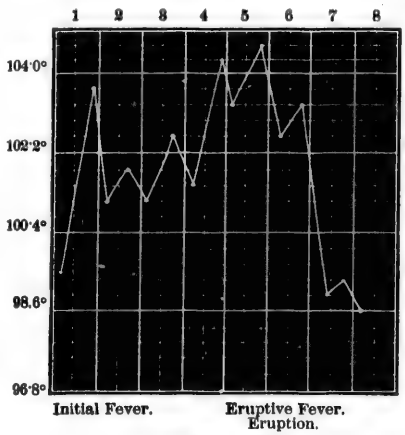


CHART X.—Measles (Strümpell).

Eruption. — Usually on the fourth day, when the fever and general symptoms have reached their height, the rash appears upon the cheeks or forehead in the form of small red papules, which increase in size and spread over the neck and thorax. When the eruption becomes well developed the face is swollen and covered with reddish blotches, which often have rounded or crescentic outlines. Here and there is an intervening portion of unaffected skin. At this stage the cervical lymph-glands may be slightly swollen and sore. The papules can now be felt with the finger. Sometimes they are quite shotty, but do not extend deep into the skin. On the trunk and extremities the swelling of the skin is not so noticeable, the color of the rash not so intense and often less uniform. The mottled blotchy character of the rash appears most clearly on the chest or the abdomen. The rash is hyperæmic and disappears on pressure, but in the more malignant cases it may become petechial. The general symptoms do not abate with the occurrence of the eruption. They persist until the end of the fifth or the sixth day, when in the majority of the cases all the symptoms become mitigated. Among the peculiarities of the rash may be mentioned the development of numerous miliary vesicles and the occurrence of petechiæ, which are seen occasionally even in cases of moderate severity.

Desquamation.—After persisting for two or three days the rash gradually fades and desquamation occurs in the form of very fine branny scales, which may be difficult to see and are wholly unlike the coarse exfoliation in scarlet fever.

The catarrhal symptoms gradually disappear and convalescence is rapidly established.

In epidemics of measles atypical cases are common. The rash may appear early, within thirty-six hours of the onset of the symptoms; or, on the other hand, it may be delayed until the sixth day. As in other exanthems, when many cases occur in a household, one of the children may

have all the initial symptoms and "sicken for the disease," as it is said, but no eruption appear.

The most serious variety of measles is that in which hæmorrhages occur—the *morbilli hæmorrhagici*. In general practice these cases are very uncommon. Occasionally in institutions, particularly when the hygienic surroundings are bad, one or two cases develop during an epidemic. It has been frequently seen in camps and when the disease is freshly imported into a native population, as in the Fiji Islands. During the civil war, as shown by Smart's statistics, some cases occurred.

In this form the disease sets in with much greater intensity, the rash becomes petechial, hæmorrhages occur from the mucous membranes, the constitutional depression is very great, and death occurs early from toxæmia.

Complications and Sequelæ.—These are met with chiefly in the respiratory system. The danger comes from the existing bronchitis, which is apt to extend into the smaller tubes and lead to collapse and broncho-pneumonia. When limited in extent this causes only aggravation of the cough and persistence of the fever (symptoms which gradually abate), and convalescence is rapid; but in debilitated children, more particularly in institutions and among the lower classes, this complication is extremely grave and is responsible for the high death-rate from measles in the community. In some instances the clinical picture is that of a suffocative catarrh, the result of a wide-spread involvement of the smaller tubes. The description of the condition will be found under the section Broncho-pneumonia. Lobar pneumonia is less common and perhaps less dangerous.

Laryngitis is not uncommon: the voice becomes husky and the cough croupy in character. Œdema of the glottis is very rare. Pseudo-membranous inflammation of the pharynx and larynx may occur and prove fatal. In debilitated infants severe stomatitis or even *cancrum oris* may develop.

Catarrhal inflammation of the middle ear is not very uncommon, and may proceed to suppuration and to perforation of the drum. The conjunctival catarrh rarely leads to further trouble, though occasionally the inflammation becomes purulent.

Intestinal catarrh is common in some epidemics, and there may be the symptoms of acute colitis.

Nephritis is an exceedingly rare complication.

Of the sequelæ of measles, tuberculosis is the most important—either an involvement of the bronchial glands, a miliary tuberculosis, or a tuberculous broncho-pneumonia.

Among the rarer sequelæ of measles are paralyses. Hemiplegia is very rare, but cases of paraplegia have been described. Thomas Barlow *

* Medico-Chirurgical Society's Transactions, 1887.

reports a fatal case in which the symptoms occurred early, the paralysis extended rapidly and involved the upper limbs, and death took place on the eleventh day. Marked vascular changes were found in the gray matter of the spinal cord, and were believed to depend on an early disseminated myelitis. Examination of the peripheral nerves was not made. Similar cases are met with in the literature, and they probably come under the division of the post-febrile polyneuritis, though of course it is not impossible that some of them, such as Barlow's case, may be due to a rapidly ascending myelitis.

Diagnosis.—From scarlet fever, with which it is most likely to be confounded, measles is distinguished by the longer initial stage with characteristic symptoms, and the blotchy irregular character of the rash, which is so unlike the diffuse uniform erythema of scarlet fever. Occasionally in measles, when the throat is very sore and the eruption pretty diffuse, there may at first be difficulty in determining which disease is present, but a few days should suffice to make the diagnosis clear. It may be extremely difficult to distinguish from rötheln. I have more than once known practitioners of large experience unable to agree upon a diagnosis. The shorter prodromal stage, the slighter fever in many cases, are perhaps the most important features. It is difficult to speak definitely about the distinctions in the rash, though perhaps the more uniform distribution and the absence of the crescentic arrangement are more constant in rötheln.

The conditions under which measles may be mistaken for small-pox have already been described. Of drug eruptions, that induced by copaiba is very like measles, but is readily distinguished by the absence of fever and catarrh.

Prognosis.—The mortality bills of large cities show what a serious disease measles is in a community. Among the eruptive fevers it ranks third in the death-rate. The mortality from the disease itself is not high, but the pulmonary complications render it one of the most serious of the diseases of children.

In some epidemics the disease is of great severity. In institutions and in armies the death-rate is often high. The fever itself is rarely a source of danger. The extension of the catarrhal symptoms to the finer tubes is the most serious indication.

Treatment.—Confinement to bed in a well-ventilated room and a milk diet are the only measures necessary in cases of uncomplicated measles. The fever rarely reaches a dangerous height. If it does it may be lowered by sponging or by the tepid bath gradually reduced. If the rash does not come out well, warm drinks and a hot bath will hasten its maturation. The bowels should be freely opened. If the cough is distressing, paregoric and a mixture of ipecacuanha wine and squills should be given. The patient should be kept in bed for a few days after the fever subsides. During desquamation the skin should be oiled daily,

and warm baths given to facilitate the process. The convalescence from measles is the most important stage of the disease. Watchfulness and care may prevent serious pulmonary complications. The frequency with which the mothers of children with simple or tuberculous bronchopneumonia tell us that "the child caught cold after measles," and the contemplation of the mortality bills should make us extremely careful in our management of this affection.

IX. RUBELLA (*Rötheln, German Measles*).

This exanthem has also the names of *rubeola notha*, or epidemic roseola, and, as it is supposed to present features common to both, has been also known as hybrid measles or hybrid scarlet fever. It is now generally regarded, however, as a separate and distinct affection.

Etiology.—It is propagated by contagion and spreads with great rapidity. It frequently attacks adults, and the occurrence of either measles or scarlet fever in childhood is no protection against it. The epidemics of it are often very extensive.

Symptoms.—These are usually mild, and it is altogether a less serious affection than measles. Very exceptionally, as in the epidemics studied by Cheadle, the symptoms are severe.

The stage of incubation ranges from ten to twelve days.

In the stage of invasion there are chilliness, headache, pains in the back and legs, and coryza. There may be very slight fever. In 30 per cent of Edwards's cases the temperature did not rise above 100°. The duration of this stage is somewhat variable. The rash usually appears on the first day, some writers say on the second, and others again give the duration of the stage of invasion as three days. Griffith places it at two days. The eruption comes out first on the face, then on the chest, and gradually extends so that within twenty-four hours it is scattered over the whole body. It may be the first symptom noted by the mother. The eruption consists of a number of round or oval, slightly raised spots, pinkish-red in color, usually discrete, but sometimes confluent.

The color of the rash is somewhat brighter than in measles. The patches are less distinctly crescentic. After persisting for two or three days (sometimes longer), it gradually fades and there is a slight furfuraceous desquamation. The rash persists as a rule longer than in scarlet fever or measles, and the skin is slightly stained after it. The lymphatic glands of the neck are frequently swollen, and, when the eruption is very intense and diffuse, the lymph-glands in the other parts of the body.

There are no special complications. The disease usually progresses favorably; but in rare instances, as in those reported by Cheadle, the symptoms are of greater severity. Albuminuria may occur and even

nephritis. Pneumonia and colitis have been present in some epidemics. Icterus has been seen.

Diagnosis.—The mildness of the case, the slightness of the prodromal symptoms, the mildness or the absence of the fever, the more diffuse character of the rash, its rose-red color, and the early enlargement of the cervical glands, are the chief points of distinction between rötheln and measles.

The treatment is that of a simple febrile affection. It is well to keep the child in bed, though this may be difficult, as the patient rarely feels ill.

X. EPIDEMIC PAROTITIS (*Mumps*).

Definition.—An infectious disease, characterised by inflammation of the parotid gland. The testes in males and the ovaries and breasts in females are sometimes involved.

Etiology.—The nature of the virus is unknown. It is probably a micro-organism, and a *bacillus parotidis* has been described.

The affection has all the characters of an epidemic disease. It is said to be endemic in certain localities, and probably is so in large centres of population. At certain seasons, particularly in the spring and autumn months, the number of cases increases rapidly. It is met most frequently in childhood and adolescence. Very young infants and adults are seldom attacked. Males are somewhat more frequently affected than females. In institutions and schools the disease has been known to attack over 90 per cent of all the children. It may be curiously localised in a city or district. The disease is contagious and spreads from patient to patient.

A remarkable idiopathic, non-specific parotitis may follow injury or disease of the abdominal or pelvic organs. Stephen Paget* has collected 101 cases of this kind, the majority of which were not associated with septic processes.

Symptoms.—The period of incubation is from two to three weeks, and there are rarely any symptoms during this stage. The invasion is marked by fever, which is usually slight, rarely rising above 101°, but in exceptionally severe cases going up to 103° or 104°. The child complains of pain just below the ear on one side. Here a slight swelling is noticed, which increases gradually, until, within forty-eight hours, there is great enlargement of the neck and side of the cheek. The swelling passes forward in front of the ear, and back beneath the sterno-cleido muscle. The other side usually becomes affected within a day or two. The submaxillary glands may also be involved. The greatest inconvenience is experienced in taking food, for the patient is unable to open the mouth, and

* British Medical Journal, March 19, 1887.

even speech and deglutition become difficult. There may be an increase in the secretion of the saliva, but the reverse is sometimes the case. There is seldom great pain, but, instead, an unpleasant feeling of tension and tightness. There may be earache and slight impairment of hearing.

After persisting for from seven to ten days, the swelling gradually subsides and the child rapidly regains his strength and health. Relapse rarely if ever occurs.

Occasionally the disease is very severe and characterised by high fever, delirium, and great prostration. The patient may even lapse into a typhoid condition.

One of the most remarkable features of the disease is a tendency to involvement of the testes. This most frequently occurs after the affection of the salivary glands has subsided. The swelling may be great, and occasionally effusion takes place into the tunica vaginalis. The orchitis is in some instances unilateral, involving the right testicle. The inflammation increases for three or four days, and resolution takes place gradually. Occasionally there may be a muco-purulent discharge. In severe cases atrophy may follow. Orchitis is rarely seen before puberty.

A vulvo-vaginitis sometimes occurs in girls, and the breasts may become enlarged and tender. Involvement of the ovaries is rare.

Complications and Sequelæ.—Of these the cerebral affections are perhaps the most serious. As already mentioned, there may be delirium and high fever. In rare instances meningitis has been found. Hemiplegia and coma may also occur. A majority of the fatal cases are associated with meningeal symptoms. These, of course, are very rare in comparison with the frequency of the disease; yet, in the Index Catalogue, under this caption, there are six fatal cases mentioned. In some epidemics the cerebral complications are much more marked than in others. Acute mania has occurred, and there are instances on record of insanity following the disease.

Arthritis is an occasional complication. Albuminuria, with convulsions, has been described. Fatal cases have occurred from acute uræmia.

Suppuration of the gland is an extremely rare complication in genuine idiopathic mumps. Gangrene has occasionally occurred. The special senses may be seriously involved. Many cases of deafness have been described in connection with or following mumps. The deafness, unfortunately, may be permanent. Affections of the eye are rare, but atrophy of the optic nerve has been described.

The diagnosis of the disease is usually easy. The position of the swelling in front of and below the ear and the elevation of the lobe on the affected side definitely fix the locality of the swelling. In children inflammation of the parotid, apart from ordinary mumps, is excessively rare.

Treatment.—It is well to keep the patient in bed during the height of the disease. The bowels should be freely opened, and the patient given a light liquid diet. No medicine is required unless the fever is high, in

which case aconite may be given. Cold compresses may be placed on the gland, but children, as a rule, prefer hot applications. A pad of cotton wadding covered with oiled silk is the best application. Suppuration is almost unknown, and need not be dreaded, even though the gland become very tense. Should redness and tenderness develop, leeches may be used. With delirium and head symptoms the ice-cap may be applied. In a robust subject, unless the signs of constitutional depression are extreme, a free venesection may do good. For the orchitis, rest, with support and protection of the swollen gland with cotton-wool, is usually sufficient.

XI. WHOOPING-COUGH.

Definition.—A specific affection characterised by convulsive cough and a long-drawn inspiration, during which the "whoop" is produced.

Etiology.—The disease occurs in epidemic form, but sporadic cases appear in a community from time to time. It is directly contagious from person to person; but dwelling-rooms, houses, school-rooms, and other localities may be infected by a sick child. It is, however, in this way less infectious than other diseases, and is probably most often taken by direct contact. The nature of the virus is still doubtful, many organisms having been described in the sputum. The observations of Afanassjew in 1887 have been the most satisfactory. He has cultivated a short bacillus, which grows with well-marked characters, and, when inoculated into the trachea of animals, produces a catarrhal condition of the mucous membrane. Cornil and Babes* conclude that the organism has not characteristics sufficiently pronounced, or an influence on animals sufficiently characteristic, to enable us to say that it is specific. Epidemics prevail for two or three months, usually during the winter and spring, and have a curious relation to other diseases, often preceding or following measles, less frequently scarlet fever.

Children between the first and second dentition are commonly affected. Sucklings are, however, not exempt, and I have seen very severe attacks in infants under six weeks. It is stated that girls are more subject to the disease than boys. Adults and old people are sometimes attacked, and in the aged it may be a very serious affection. Many persons possess immunity against the disease, and, though frequently exposed, escape. Delicate anæmic children with nasal or bronchial catarrh are more subject to the disease than others. According to the United States Census Reports, the disease is more than twice as fatal in the negro race than in others.

Morbid Anatomy.—Whooping-cough itself has no special pathological changes. In fatal cases pulmonary complications, particularly broncho-pneumonia, are usually present. Collapse and compensatory em-

* Les Bactéries, 1890.

physema, vesicular and interstitial, are found, and the tracheal and bronchial glands are enlarged.

Symptoms.—Catarrhal and paroxysmal stages can be recognized. There is a variable period of incubation of from seven to ten days. In the *catarrhal stage* the child has the symptoms of an ordinary cold, which may begin with slight fever, running at the nose, injection of the eyes, and a bronchial cough, usually dry and sometimes giving indications of a spasmodic character. The fever is usually not high, and slight attention is paid to the symptoms, which are thought to be those of an ordinary cold. After lasting for a week or ten days, instead of subsiding, the cough becomes worse and more convulsive in character.

The *paroxysmal stage*, marked by the characteristic cough, dates from the first appearance of the "whoop." The fit begins with a series of from fifteen to twenty short coughs of increasing intensity, and then with a deep inspiration the air is drawn into the lungs, making the "whoop," which may be heard at a distance and from which the disease takes its name. This loud inspiratory sound may sometimes precede the series of spasmodic expiratory efforts. Several coughing-fits may succeed each other until a tenacious mucus is expectorated. This may be small in amount, but after a series of coughing-fits a considerable quantity may be expectorated. Not infrequently it is brought up by vomiting or by a combination of cough and regurgitation. There may be only four or five of these attacks in the day, or in severe cases they may recur every half-hour. During the attack the thorax is very strongly compressed by the powerful expiratory efforts, and, as very little air passes in through the glottis, there are signs of defective aëration of the blood; the face becomes swollen and congested, the veins are prominent, the eyeballs protrude, and the conjunctivæ become deeply engorged. Suffocation indeed seems imminent, when with a deep crowing inspiration air enters the lungs and the color is quickly restored. Children are usually terrified at the onset, and run at once to the mother or nurse to be supported during the attack. Few diseases are more painful to witness. In severe paroxysms vomiting is frequent and the sphincters may be opened.

An ulcer under the tongue is a very common event, and was thought at one time to be the cause of the disease.

During the attack, if the chest be examined, the resonance is defective in the expiratory stage, full and clear during the deep, crowing inspiration; but on auscultation during the latter there may be no vesicular murmur heard, owing to the slowness with which the air passes the narrowed glottis. Bronchial râles are occasionally heard.

Among circumstances which precipitate an attack are emotion, such as crying, and any irritation about the throat. Even the act of swallowing sometimes seems sufficient. In a close dusty atmosphere the coughing-fits are more frequent. After lasting for three or four weeks the attacks

become lighter and finally cease. In cases of ordinary severity the course of the disease is rarely under six weeks.

The complications and sequelæ of whooping-cough are important. During the extensive venous congestion hæmorrhages are very apt to occur in the form of petechiæ, particularly about the forehead, ecchymosis of the conjunctivæ, epistaxis, and occasionally hæmoptysis. Hæmorrhage from the bowels is rare. During the paroxysm convulsions may occur, due perhaps to the extreme engorgement of the cerebral cortex. Very rarely hemiplegia or monoplegia follows. Sudden death has been caused by extensive subdural hæmorrhage. Whooping-cough must be regarded as a very unusual cause of cerebral palsy in children. It was associated with three cases of my series of one hundred and twenty cases, but in none of them did the hemiplegia come on during the paroxysm, as in a case reported by S. West.

The persistent vomiting may induce marked anæmia and wasting. The pulmonary complications which follow whooping-cough are extremely serious. During the severe coughing-spells interstital emphysema may be induced, more rarely pneumothorax. I saw one instance in which rupture occurred, evidently near the root of the lung, and the air passed along the trachea and reached the subcutaneous tissues of the neck, a condition which has been known to become general. Broncho-pneumonia, with its accompanying collapse, is the most frequent pulmonary complication and carries off a large number of children. It may be simple, but in a considerable proportion of the cases the process is tuberculous. Pleurisy is sometimes met with and occasionally lobar pneumonia. Enlargement of the bronchial glands is very common in whooping-cough and has been thought to cause the disease. It may sometimes be sufficient to produce dulness upon the manubrium. The heart stands the strain of whooping-cough remarkably well. During the spasm the radial pulse is small, the right heart engorged, and during and after the attack the cardiac action is very much disturbed. It is difficult to determine whether serious damage ever results. Possibly some of the cases of severe valvular disease in children who have had neither rheumatism nor scarlet fever may be attributed to the terrible heart strain during a prolonged attack of whooping-cough. Renal complications are very uncommon. Sugar is occasionally found in the urine.

Diagnosis.—So distinctive is the "whoop" of the disease that the diagnosis is very easy; but occasionally there are doubtful cases, particularly during epidemics, in which a series of expiratory coughs occurs without any inspiratory crow.

Prognosis.—Taken with its complications, whooping-cough must be regarded as a very fatal affection. According to Dolan it ranks third among the fatal diseases of children in England, where the death-rate per million from this disease is five thousand annually. The younger the infant the greater is the probability of serious complications. The

deaths are chiefly among children of the poor and among delicate infants.

Treatment.—Parents should be warned of the serious nature of whooping-cough, the gravity of which is scarcely appreciated by the public. Particular care should be taken that children suspected of the disease are not sent to the public schools or exposed in any way so that other children can become contaminated. There is more reprehensible neglect in connection with this than with any other disease. The medicinal treatment of whooping-cough is most unsatisfactory. Like other infectious disorders it runs its course practically uninfluenced in a majority of cases by drugs. In the catarrhal stage when there is fever the child should be in bed and a saline fever mixture administered. If the cough is distressing, ipecacuanha wine and paregoric may be given. For the paroxysmal stage a suspiciously long list of remedies has been recommended, twenty-two in one popular text-book on therapeutics. If the disease is due, as seems probable, to a germ growing upon and irritating the bronchial mucosa, a germicidal plan of treatment seems highly rational and persistent attempts should be made to discover a suitable remedy. Quinine placed upon the tongue; resorcin in one-per-cent solutions, swabbed frequently on the throat; two or three grains of iodoform to an ounce of starch powder; a spray of carbolic acid—have all been warmly recommended. J. Lewis Smith advises the use of the steam atomizer with a solution of carbolic acid, chloride of potassium and bromide of potassium in glycerin. Jacobi regards belladonna as the most satisfactory remedy. He gives it in full doses, as much as one sixth of a grain of the root or the extract to a child of six or eight months three times a day. It should be given in sufficient doses to produce the cutaneous flush. For the nervous element in the disease antipyrin has been used with apparent success.

After the severity of the attack has mitigated and convalescence has begun, the child should be watched with the greatest care. It is just at this period that the fatal broncho-pneumonias are apt to develop. The cough sometimes persists for months and the child remains weak and delicate. Change of air should be tried. Such a patient should be fed with care, and given tonics and cod-liver oil.

XII. INFLUENZA (*La Grippe*).

Definition.—An infectious disease characterised by great prostration and often catarrh of the mucous membranes, particularly the respiratory and gastro-intestinal. There is a marked liability to serious complications, particularly pneumonia.

Epidemics appear at intervals and spread with extraordinary rapidity, so that in a few weeks an entire continent may be involved. The dis-

ease has been known for several centuries, and there have been within the past fifty years several extensive outbreaks, notably those of 1833, 1847–'48, and the recent outbreak in 1889–'90. Many of the epidemics have started in Russia, hence the name Russian fever. In October of 1889 it prevailed extensively in St. Petersburg. During November and December it spread to Germany, France, and western Europe, appearing in London about the end of December. Cases appeared in this country about Christmas, and the disease rapidly became epidemic.

Etiology.—The conditions which favor its development and rapid spread are unknown, and the exhaustive literature of the past year has not brought us nearer a solution of the problem. It appears to be independent of meteorological conditions. While some authorities hold that the affection is due to a miasmatic material in the atmosphere, others probably more correctly hold that it is due to a specific virus of the most intense infectiveness. Like other rapidly spreading diseases, it is conveyed along lines of travel. The bacteriological examinations which have been made in large numbers of cases leave us still in doubt, and the varied character of the germs found by reliable observers indicates that the true virus has not yet been detected. The pus organisms and the *diplococcus pneumoniæ* have been found oftenest, but these are widespread organisms and are probably not associated in a causative manner with the disease.

Morbid Anatomy.—Uncomplicated cases recover. In the delicate and aged alone do we see fatal results, and then only from the intensity of the fever or the profound depression. Injection and swelling of the pharyngeal and laryngeal mucosa, bronchitis, and a catarrhal condition of the stomach and intestines may be present.

The complications are very varied. Severe bronchitis, lobar and lobular pneumonia, and nephritis may exist.

Symptoms.—In many cases the attack closely resembles an ordinary catarrh with slight fever, dryness and swelling of the nasal mucosa, and then increase in the secretion. In the severer cases the coryza is subsidiary or absent, and the symptoms are those of an infection of varying grades of severity. A striking feature is the severe nervous manifestation at the outset, the headache, pain in the back and legs, and a general soreness as if bruised or beaten. With the exception of dengue and small-pox there is no affection in which these symptoms are more pronounced. Delirium may be marked. Associated with these is a prostration and cardiac weakness out of proportion to the intensity of the fever, and sometimes very alarming. The pulse is feeble, small, and intermittent. Death may result directly from heart-failure, as in cases mentioned by Wilks.

Serious nervous complications are marked *delirium* and *meningitis*, the latter usually in association with pneumonia. Bristowe has reported several cases of *abscess of the brain* following influenza. Peripheral neuritis was not very uncommon in the last epidemic. *Mental disorders* are

not infrequent. Inaptitude for mental exertion, depression of spirits, even insanity, may follow an attack.

Affections of the respiratory organs are the most serious. Many cases present an intense *bronchitis*, involving the large and small tubes and coming on with high fever, sometimes with delirium. An intense general bronchitis was common during the recent epidemic. In children it may be complicated with broncho-pneumonia. By far the most serious and fatal complication is *pneumonia*, which may follow the bronchitis, or set in with well-characterised symptoms. Sometimes the symptoms may at first be obscure and the pneumonia atypical. Thus, after an initial rigor, with some dyspnœa and high fever, the local signs may be obscure and it may not be until the third or fourth day, or even later, that the physical signs of a pneumonia are detected. The sputa may not be rusty until the fourth or fifth day. The crisis may be deferred or the defervescence may be by lysis. A considerable proportion of the cases, however, run a normal course. So far as I could see, there was nothing special or peculiar in the pneumonia; all the anomalies which have been mentioned as occurring in influenza are found in any large series of cases. Abscess of the lung may follow. Pleurisy is not an uncommon complication, and empyema may develop.

The gastro-intestinal symptoms may be marked; thus, with the initial fever, there may be nausea and vomiting. Diarrhœa is not uncommon; indeed, the brunt of the catarrhal process may fall upon the gastro-intestinal mucosa.

The *diagnosis* of the disease offers no difficulties when it occurs in epidemic form. Coryza is not always present, and the symptoms may be those of general fever with great prostration. In other instances the bronchitis may be an important feature. The severe prostration, fever, delirium, with the initial bronchitis, and occasionally epistaxis, may lead to the diagnosis of typhoid fever. The complications are, as a rule, readily recognised, though at first the symptoms of the pneumonia may be somewhat indefinite.

Treatment.—In every case the disease should be regarded as serious, and the patient should be confined to bed until the fever has completely disappeared. In this way alone can serious complications be avoided. From the outset the treatment should be supporting, and the patient should be carefully fed and well nursed. The bowels should be opened by a dose of calomel or a saline draught. At night ten grains of Dover's powder may be given. At the onset a warm bath is sometimes grateful in relieving the pain in the back and limbs, but great care should be taken to have the bed well warmed, and the patient should be given after it a drink of hot lemonade. If the fever is high and there is delirium, small doses of antipyrin may be given and an ice-cap applied to the head. The medicinal antipyretics should be used with caution, as profound prostration sometimes develops in these cases. Too much stress should not be

laid upon the mental features. Delirium may be marked even with slight fever. In the cases with great cardiac weakness stimulants should be given freely, and during convalescence strychnia in full doses.

The intense bronchitis, pneumonia, and other complications should receive their appropriate treatment. The convalescence requires careful management, and it may be weeks or months before the patient is restored to full health. A good nutritious diet, change of air, and pleasant surroundings are essential. The depression of spirits following this disease is one of its most unpleasant and obstinate features.

XIII. DENGUE.

Definition.—An acute infectious disease of tropical and subtropical regions, characterised by febrile paroxysms, pains in the joints and muscles, and sometimes a cutaneous rash.

The disease was first noted in Java toward the close of the last century, and it was probably described by Rush in 1780. During this century many epidemics of it have been reported, particularly in India, Africa, and the southern United States. S. H. Dickson gave the most satisfactory account of the disease as it appeared in Charleston in 1828. Since that time there have been three or four wide-spread epidemics, confined chiefly to the Gulf States and rarely extending beyond the 32nd parallel.

Etiology.—Many observers regard it as contagious, and Dickson mentions in the history of his own household that during the epidemic of 1828 all were attacked, whereas in the epidemic of 1850 he and the cook (the only ones remaining in his household of those who composed it in 1828) alone escaped. The question can scarcely yet be considered settled. The disease spreads from place to place, and is conveyed by ships and along railroads. It is remarkable among epidemics as practically affecting all members in a community who have not been protected by a previous attack. Matas, in his excellent account,* states that one attack does not protect from subsequent infection. It attacks all races equally. The disease is stated to attack animals.

McLoughlin, of Texas, has found in the blood of patients a micrococcus, which he regards as the special agent and has been able to cultivate. The slides which he kindly sent me show a streptococcus-like organism, but it is impossible yet to speak definitely as to the relations which it bears to the disease. If it be true that animals are subject to the affection, the subject could be conclusively worked out during the next epidemic. Some writers have held that dengue is only a modified form of yellow fever. It has in some instances preceded the development of this disease.

* Keating's Encyclopedia of Diseases of Children, vol. i.

As the disease is never fatal, no observations have been made upon its pathological anatomy.

Symptoms.—The period of incubation is from three to five days, during which the patient feels well. The attack sets in suddenly with headache, chilly feelings, and intense aching pains in the joints and muscles. The fever rises gradually and may reach as high as 106° or 107°. The pulse is rapid and there are the other phenomena associated with acute fever—loss of appetite, coated tongue, slight nocturnal delirium, and concentrated urine. In the initial stage there may be an erythematous rash. In a majority of the cases the pains in the muscles, joints and bones are of a most aggravated character, and the patients speak of them as of a boring or breaking character, hence the popular name "break-bone fever." The large and small joints are affected, sometimes in succession, and they become swollen, red, and painful. The pains shift about, and in some cases cutaneous hyperæsthesia has been noted. In some instances there is a tendency to hæmorrhage, from either the nose, lungs, stomach, or bowels. Eugene Foster speaks of having seen black vomit, similar to that of yellow fever, and in three instances alarming hæmorrhage from the bowels, which in one case persisted for three months and caused death.

The fever gradually reaches its height by the third or fourth day, and the patient enters upon the apyretic period, which may last from two to four days, and in which he feels prostrated and stiff. At this time, in a large number of cases, an eruption is common which, judging from the description, has nothing distinctive, being at times macular, like measles, at others, diffuse and scarlatiniform, or papular, or lichen-like. In other instances the rash has been described as urticarial, or even vesicular. A second paroxysm of fever then occurs, and the pains return. Certain writers describe inflammation and hyperæmia of the mucous membrane of the nose, mouth, and pharynx. Enlargement of the lymph-glands is not uncommon, and may persist for weeks after the disappearance of the fever. Convalescence is often protracted, and there is a degree of mental and physical prostration out of all proportion to the severity of the primary attack. By far the most distressing symptom is the pain, which all who have experienced the disease speak of as agonising and intolerable, and more severe than that experienced in any other acute fever.

Complications are rare. Insomnia and occasionally delirium, resembling somewhat the alcoholic form, have been observed. A relapse may occur even as late as two weeks. Briefly, the course of the disease may be described as consisting of a febrile paroxysm of three or four days; a remission of variable duration, which may be wanting; and a second paroxysm of about three days. The average duration of a moderate attack is from seven to eight days.

The *diagnosis* of the disease rarely offers any special difficulties, prevailing as it does in epidemic form, and attacking all classes indiscriminately. Isolated cases might be mistaken at first for acute rheumatism.

Southern physicians say that occasionally yellow fever and dengue may be confounded.

Treatment.—This is entirely symptomatic. Quinine is stated to be a prophylactic, but on insufficient grounds. Hydrotherapy may be employed to reduce the fever. The salicylates or antipyrin may be tried for the pains, which usually, however, require opium. During convalescence iodide of potassium is recommended for the arthritic pains, and tonics are indicated.

XIV. CEREBRO-SPINAL MENINGITIS.

Definition.—A specific infectious disease, occurring sporadically and in epidemics, characterised by inflammation of the cerebro-spinal meninges and a clinical course of great irregularity.

The affection is known by the names of malignant purpuric fever, petechial fever, and spotted fever.

Etiology.—Since its recognition in Geneva in the early part of this century, numerous epidemics have been described in Europe and in America, the full details of which are to be found in Stillé's elaborate article.* In Europe it is remarkable with what frequency the disease has occurred in garrisons. In this country the disease was first seen in Massachusetts in 1806, since which date there have been epidemics in various localities at irregular intervals.

During the civil war, according to Smart's report, comparatively few deaths were caused by this disease.

Sporadic cases occur from time to time in the larger cities and country districts on this continent. After the first epidemic in Montreal in 1873 occasional cases occurred. In Philadelphia, since its appearance in 1863, there have been cases reported every year in the mortality bills. Without autopsy the diagnosis of many of these cases is extremely doubtful; but there can be no question that the disease, though rare, still lingers. Judging from my own experience in three of the hospitals of that city, and from the fact that in five years I saw only three instances, I would regard it as very much less frequent than the reports of the Health Office would seem to indicate.

The disease has broken out simultaneously in regions far distant from each other.

The epidemics have occurred most frequently in winter and spring. Neither soil nor locality has any special influence. The concentration of individuals, as in large barracks, seems to be specially favorable.

Children are much more susceptible to the disease than adults, though the susceptibility has differed in different epidemics. In certain places

* System of Medicine, Philadelphia, vol. i, 1885.

children alone have been affected; in others the disease has been chiefly among adults. It attacks males and females alike.

Certain epidemics have been most prevalent in country districts. In 1873 the disease prevailed along the valley of the Ottawa, in villages and country places, much more severely than in the cities of Montreal and Ottawa.

Over-exertion, prolonged marching in the heat, depressing mental or bodily surroundings, and the misery and squalor of the large tenement-houses in cities are predisposing causes.

The disease is not directly contagious; it is probably not transmitted by clothing or the excretions.

The nature of the virus is as yet unknown. In the meningeal exudation there is now found in many cases the lance-shaped coccus, similar in all respects to the pneumococcus. In other instances this microbe has been associated with the ordinary pus organisms. Cornil and Babes conclude that cerebro-spinal meningitis may be caused by several different, often associated, forms of micro-organisms, of which the lance-shaped coccus of Pasteur is the most common.

Morbid Anatomy.—In malignant cases there may be no characteristic changes, for the patient may die before exudation occurs. In well-marked cases the meninges of the brain and cord are inflamed. The following abstract of one of the Montreal cases, in which death occurred about the fifth day, gives a good idea of the condition in this disease: The brain contained an excessive amount of blood. The dural sinuses and all the veins and arteries were engorged. Some of the veins of the pia were as large as goose-quills. On the cortex there was much lymph beneath the arachnoid on either side of the longitudinal fissure—more on the right than on the left hemisphere. At the base there was a purulent exudate about the chiasma and inner parts of the Sylvian fissure, but none on the pons or medulla. There was no lymph in the course of the middle cerebral arteries. The ventricles contained serous exudate; the walls were not softened. The gray matter of the brain was deeply congested, but presented neither hæmorrhages, spots, nor softening. In the spinal cord the veins of the pia were engorged. On the posterior surface, from the cervical enlargement to the cauda equina, was a thick layer of grayish-yellow, lympho-purulent exudation, which in places produced irregular bulging of the arachnoid membrane. There were no changes in the thoracic or abdominal viscera. This picture corresponds closely with that presented by five other cases which I have examined. In one case, however, the amount of exudation on the hemispheres was large, and the convolutions were covered with a thick creamy pus. Foci of hæmorrhage and of encephalitis occur in some cases. The formation of abscess has been occasionally described. The involvement of the ventricles is less than in tuberculous meningitis. In the cases which I have seen the exudation, as is usual in the secondary meningeal inflammations, was most

abundant on the cortex. The exudation may extend along the lymph-sheaths of the cranial nerves, particularly the auditory and optic. In long-standing cases the inflammatory processes appear more chronic. There are thickening and adhesion of the membranes, areas of cortical softening or of atrophy, and, in some instances, hydrocephalus. The changes in the other organs are those associated with fever. In the malignant cases there may be hæmorrhages into the skin and on the serous membranes. Pneumonia, pleurisy, endocarditis, dysentery and nephritis have been described. The spleen varies in size according to the period of the disease at which death has occurred. When the fever has been intense it is enlarged.

Symptoms.—Cases differ remarkably in their characters. Many different forms have been described. These are perhaps best grouped into three classes:

1. **Malignant Form.**—This fulminant or apoplectic type occurs with variable frequency in epidemics. It may occur sporadically. The onset is sudden, usually with violent chills, headache, somnolence, spasms in the muscles, great depression, moderate elevation of temperature, and feeble pulse, which may fall to fifty or sixty in the minute. Usually a purpuric rash develops. In a Philadelphia case in 1888 a young girl, apparently quite well, died within twenty hours of this form. There are cases on record in which death has occurred within a shorter time. Stillé tells of a child of five years, in whom death occurred after an illness of ten hours; and refers to a case reported by Gordon, in which the entire duration of the illness was only five hours.

2. **Ordinary Form.**—The stage of incubation is not known. The disease usually sets in suddenly. There may be premonitory symptoms: headache, pains in the back, and loss of appetite. More commonly, the onset is with headache, severe chill, and vomiting. The temperature rises to 101° or 102°. The pulse is full and strong. An early and important symptom is a painful stiffness of the muscles of the neck. The headache increases, and there are photophobia and great sensitiveness to noises. Children become very irritable and restless. In severe cases the contraction of the muscles of the neck sets in early, the head is drawn back, and, when the muscles of the back are also involved, there is opisthotonos. The pains in the back and in the limbs may be very severe. The motor symptoms are most characteristic. Tremor of the muscles may be present, with tonic or clonic spasms in the arms or legs. Rigidity of the muscles of the back or neck is very common, and the patient lies with the body stiff and the head drawn so far back that the occiput may be between the shoulder-blades. Except in early childhood convulsions are not common. Strabismus is a frequent and important symptom. Spasm of the muscles of the face may also occur. Cases have been described in which the general rigidity and stiffness was such that the body could be moved like a statue. Paralysis of the trunk mus-

cles is rare, but paralysis of the muscles of the eye and the face is not uncommon.

Of sensory symptoms, headache is the most dominant and persists from the outset. It is chiefly in the back of the head, and the pain extends into the neck and back. There may be great sensitiveness along the spine, and in many cases there is marked hyperæsthesia.

The psychical symptoms are marked. Delirium occurs at the outset, occasionally of a furious and maniacal kind. The patient may display marked erotic symptoms. The delirium gives place in a few days to stupor, which, as the effusion increases, deepens to coma.

The temperature is irregular and variable. Remissions occur frequently, and there is no uniform or typical curve during the disease. In some instances there has been little or no fever. In other cases the temperature may reach 105° or 106°, or, before death, 108°. The pulse may be very rapid in children; in adults it is at first usually full and strong. In some cases it is remarkably slow, and may not be more than fifty or sixty in the minute. Sighing respirations and Cheyne-Stokes breathing are met with in some instances. Unless there is pneumonia the respirations are not often increased in frequency.

The cutaneous symptoms of the disease are important. Herpes labialis occurs with even greater frequency than in pneumonia or in intermittent fever. The petechial rash, which has given the name spotted fever to the disease, is very variable. Stillé states that of ninety-eight cases in the Philadelphia Hospital, no eruption was observed in thirty-seven. In the Montreal cases petechiæ and purple spots were common. They appear to have been more frequent in the epidemics on this continent than in Europe. The petechiæ may be numerous and cover the entire skin. An erythema or dusky mottling may be present. In some instances there have been rose-colored hyperæmic spots like the typhoid rash. Urticaria or erythema nodosum, ecthyma, pemphigus and in rare instances gangrene of the skin have been noted.

As already stated, vomiting may be a special feature at the outset; but, as a rule, it gradually subsides. In some instances, however, it persists and becomes the most serious and distressing of the symptoms. Diarrhœa is not common. The bowels are usually confined. The abdomen is not tender. In acute cases the spleen is usually enlarged.

The urine is sometimes albuminous and the quantity may be increased. Glycosuria has been noted in some instances, and in the malignant forms hæmaturia.

The course of the disease is extremely variable. Hirsch rightly states that it may range between a few hours and several months. More than half of the deaths occur within the first five days. In favorable cases, after the symptoms have persisted for five or six days, improvement is indicated by a lessening of the spasm, reduction of the fever, and a return of the intelligence. Sudden fall in the temperature is of bad omen. Con-

valescence is extremely tedious, and may be interrupted by complications and sequelæ to be noted.

3. **Anomalous Forms.**

(*a*) *Abortive Type.*—The attack sets in with great severity, but in a day or two the symptoms subside and convalescence is rapid. Strümpell would distinguish between this abortive variety, which sets in with such intensity, and the mild ambulant cases described by certain writers. He reports a case in which the meningeal symptoms set in with the greatest intensity and persisted for four days, the temperature rising to 40·9° C. On the fifth day the patient entered upon a rapid and satisfactory convalescence. In the mild cases, as distinguished from the abortive, the patients complain of headache, nausea, sensations in the back and limbs, and stiffness in the neck. There is little or no fever, and only moderate vomiting. These cases have been met with, and could be recognised only during the prevalence of an epidemic.

(*b*) *An intermittent type* has been observed in many epidemics, and is recognised by von Ziemssen and Stillé. It is characterised by exacerbations of fever, which may recur daily or every second day, or follow a curve of an intermittent or remittent character. The pyrexia resembles that of pyæmia rather than malaria.

(*c*) *Chronic Form.*—Heubner states that this is a relatively frequent form, though it does not seem to be recognised by many writers on the subject. An attack may be protracted for from two to five or even six months, and may cause the most intense marasmus. The attack consists of a series of recurrences of the fever, and may present the most complex symptomatology. It is not improbable that these protracted cases depend upon chronic hydrocephalus or abscesses of the brain. This form differs distinctly from the intermittent type. A very remarkable instance of it is described by Worthington,* in which the disease lasted for fourteen weeks.

Complications.—Pleurisy and pericarditis are not uncommon.

Pneumonia is described as frequent in certain epidemics. Immermann found, during the Erlangen epidemic, many instances of the combination of pneumonia with meningitis, but it does not seem possible to determine whether, in such instances, pneumonia is the primary disease and the meningitis secondary, or *vice versa*. The frequency with which inflammation of the meninges of the brain complicates pneumonia has already been mentioned. It is not impossible that the pneumococcus is responsible for both affections. Arthritis has been the most frequent complication in certain epidemics. Many joints are affected simultaneously, and there are swelling, pain, and exudation, sometimes serous, sometimes purulent. This was first observed by James Jackson, Sr., in the epidemic which he described.

* Canada Medical and Surgical Journal, vol. xiv.

Among the important sequelæ are those affecting the special senses. Blindness may result from optic neuritis with atrophy. Keratitis with ulceration may develop. This may also occur in the meningitis following pneumonia. Iritis is less common.

Still more serious are the ear symptoms, particularly in children. Deafness very often follows inflammation of the labyrinth; the result, no doubt, of the direct extension of the inflammation along the auditory nerve. In children this not infrequently leads to deaf-mutism. Von Ziemssen states that in the deaf and dumb institutions of Bamberg and Nuremberg, in 1874, almost all the pupils had become deaf from epidemic cerebro-spinal meningitis.

Headache may persist for months or years after an attack. Chronic hydrocephalus develops in certain instances in children. The symptoms of this are "paroxysms of severe headache, pains in the neck and extremities, vomiting, loss of consciousness, convulsions, and involuntary discharges of fæces and urine" (von Ziemssen). Von Ziemssen regards chronic hydrocephalus as by no means a rare sequela. Mental feebleness and aphasia have occasionally been noted.

Paralysis of individual cranial nerves or of the lower extremities may persist for some time. In some of these cases unquestionably there may be peripheral neuritis, as Mills suggested.

Diagnosis.—There are several affections with which cerebro-spinal meningitis is likely to be confounded:

(*a*) *Tuberculous Meningitis.*—In sporadic cases it is sometimes impossible to determine the nature of a case in the absence of local tuberculous disease. Retraction of the neck and spasms of the muscles of the arms and legs are not nearly so marked and prominent in tuberculous meningitis. Herpes also is rare, and the pulse is more irregular. There is rarely petechial eruption. When the disease is prevailing epidemically this factor is of the greatest help in the diagnosis.

(*b*) *Pneumonia.*—The meningeal complication of this disease is most commonly confined to the cerebrum. As the cortex is chiefly involved, there may be a good deal of motor spasm and tremor, but rarely is there retraction of the muscles of the neck or opisthotonos. In sporadic cases, as has been said, it may be quite impossible to decide whether the pneumonia has complicated the meningitis or the meningitis the pulmonary affection. The bacteriological examination gives no clue, as the pneumococcus is found in both situations.

(*c*) *With other Acute Infectious Diseases.*—Both typhus and typhoid present symptoms which closely simulate cerebro-spinal meningitis. On several occasions at the Montreal General Hospital cases have been sent into the ward with the diagnosis of cerebro-spinal fever. These cases showed high fever, delirium, retraction of the neck, spasm, and tremor of the muscles, and had not the post-mortem examination revealed typhoid lesions and only cerebro-spinal congestion the diagnosis would not have

been corrected. I am sure that many of the cases sent into the health offices as cerebro-spinal fever are instances of the cerebral form of typhoid.

I have already referred to the fact that the malignant form of small-pox may be mistaken for cerebro-spinal meningitis.

It could scarcely be possible to confound tetanus with this disease.

Prognosis.—Hirsch states that the mortality has ranged in various epidemics from 20 to 75 per cent. In children the death-rate is much higher than in adults. Cases with deep coma, repeated convulsions, and high fever rarely recover. The outlook in the protracted cases is not good, though Heubner gives an instance of a lad of seven, who was ill from the end of February until the end of June, with repeated recurrences, was worn to a skeleton, and yet completely recovered.

Treatment.—The high rate of mortality which has existed in most epidemics indicates the futility of the various therapeutical agents which have been recommended. When we consider the nature of the local disease and the fact that, so far as we know, simple or tuberculous cerebro-spinal meningitis is invariably fatal, we may wonder rather that recovery follows in any well-developed case.

In strong robust patients the local abstraction of blood by wet cups on the nape of the neck relieves the pain. General bloodletting is rarely indicated. Cold to the head and spine, which was used in the first epidemics by New England physicians, is of great service. A bladder of ice to the head, or an ice-cap, and the spinal ice-bag may be continuously employed. The latter is very beneficial. Judging from the beneficial effects of the general bath in typhoid with pronounced cerebro-spinal symptoms, hydrotherapy should be systematically employed if the temperature is above 102½°. In private practice the cold-pack or sponging may be substituted. If any counter-irritation is thought necessary, the skin of the back of the neck may be lightly touched with the Paquelin thermo-cautery. Blisters, which have been used so much, are of doubtful benefit and should not be employed. Of internal remedies opium may be given freely, best as morphia hypodermically. Stillé recommends either a grain of opium every hour in severe cases or every two hours in cases of moderate severity; von Ziemssen advises the hypodermic of morphia, from one third to one half grain in adults. Mercury has no special influence on meningeal inflammation. Iodide of potassium is warmly recommended by some writers. Quinine in large doses, ergot, belladonna and Calabar bean have had advocates. Bromide of potassium may be employed in the milder cases, but it is not so useful as morphia to control the spasms.

The diet should be nutritious, consisting of milk and strong broths while the fever persists. Many cases are very difficult to feed, and Heubner recommends forced alimentation with the stomach-tube. These cases seem to bear stimulants well, and whisky or brandy may be given freely when there are signs of a failing heart.

XV. DIPHTHERIA.

Definition.—A specific infectious disease, characterized by a local fibrinous exudate, usually upon a mucous membrane, and by constitutional symptoms of varying intensity. The presence of the Klebs-Loeffler bacillus may be regarded as the etiological criterion by which true diphtheria may be distinguished from other forms of pseudo-membranous inflammation.

Historical Note.—The disease was known to Aretæus and Galen. Epidemics occurred throughout the middle ages. It appeared early among the settlers of New England, and accounts are extant of epidemics in this country in the seventeenth and eighteenth centuries. Huxham and Fothergill gave excellent descriptions of the disease. An admirable account was given by Samuel Bard,* of New York, in 1770, whose essay is one of the most solid contributions made to medicine in America. It was reserved for Pierre Bretonneau, of Tours, to grasp the fact that *angina suffocativa*, "*cynanche maligna*," the "putrid" and other forms of malignant sore throat were one and the same disease, to which he gave the name "diphthérite."

Etiology.—The disease is endemic in the larger centres of population, and becomes epidemic at certain seasons of the year. It is a remarkable fact that while other contagious diseases have diminished within the past decade, diphtheria, particularly in cities, has increased. It is by no means confined to the poorer districts, but occurs in the houses of the better classes, particularly when the plumbing is defective. The disease is, however, not confined to cities. It has prevailed with great severity in country districts, in which indeed the affection seems to be specially virulent. The relation between imperfect drainage and the diphtheria poison has not yet been satisfactorily determined. Perhaps, as Thorne suggests, the faulty conditions produce sore throat of a benign character, which, as in scarlet fever, affords a soil suitable for inoculation by the diphtheria germ, when present in the air. Drains, too, he thinks may retain the virus received through the sputa and dejecta of the sick. This author states that no prevalence of diphtheria has ever been definitely traced to polluted water.

Diphtheria is a highly contagious disease, readily communicated from person to person. The poison is given off in the pharyngeal secretion and in the saliva, but not in the breath. No disease of temperate regions proves more fatal to physicians and nurses. There seems to be particular danger in the examination and swabbing of the throat, for in the gagging, coughing, and spluttering efforts the patient may cough mucus and flakes of membrane into the physician's face. The virus attaches itself to the clothing, the bedding, and the room in which the patient has lived, and

* Transactions of the American Philosophical Society, vol. i, Philadelphia, 1770.

has, in many instances, displayed great tenacity. The disease may be transmitted by inoculation. The contagion does not seem to be widely diffused in the neighborhood of the patient. At the Montreal General Hospital we rarely had cases develop in the wards adjacent to those in which there were diphtheria patients.

There is a wide-spread belief in the profession that the disease may be communicated from animals. There is in calves a contagious pseudo-membranous affection which is said to be communicable to man. Cows are not known to be affected spontaneously. In the epidemics in which the contagion has been traced to the milk, it is more probable that the virus has been accidentally mixed with it than that the cows were themselves diseased. Cats are subject to a pseudo-membranous disease, and there are many cases on record in which children appear to have caught diphtheria from them. On the other hand, I know of one case in which a cat died of angina and intense pseudo-membranous colitis, and the children who nursed it did not take the disease; and of a second case, in which a pet cat had coryza, difficult breathing, fever, and enlarged cervical glands, and here too the children were not affected. The so-called diphtheria of fowls is apparently not associated with the same germ as the human diphtheria.

Of predisposing causes *age* is one of the most important. Very young children are rarely attacked, but Jacobi states that he has seen three instances of the disease in the newly born. Between the third and the fifteenth year a large majority of the cases occur. In this period the greatest number of deaths is between the second and the fifth years. Girls are attacked in larger numbers than boys, probably because they are brought into closer contact with the sick. Adults are frequently affected. The disease is most prevalent in the cold autumn weather.

Caillé regards as special predisposing elements in children, enlarged tonsils, chronic naso-pharyngeal catarrh, carious teeth, and an unhealthy condition of the mucous membrane of the mouth and throat.

Epidemics vary in intensity. While in some the affection is mild and rarely fatal, in others it is characterized by wide extension of the membrane, and shows a special tendency to attack the larynx.

The Specific Germ.—The bacillus originally described by Klebs and more thoroughly studied by Loeffler appears to be the specific virus. It is found in the pseudo-membranes, not in the subjacent mucosa, or in the blood, or in the internal organs. It is a non-motile bacillus, varying from 2·5 to 3 μ in length, and from 0·5 to 0·8 μ in thickness. It appears as a straight or slightly bent rod with rounded ends. Irregular bizarre forms, such as rods with one or both ends swollen, are, however, not uncommon. The bacillus stains in sections or cover-glass specimens by Gram's method. It is best cultivated on blood serum and bouillon. The colonies are large, elevated, grayish-white, with an opaque centre. Welch and Abbott also state that it grows well on potato; but the growth is invisible or indicated

only by a dry thin glaze. It multiplies readily in milk. Although it forms no spores, it is a very persistent bacillus, and cultures have been made from membrane preserved for five months in a dry cloth. The cultures inoculated into the trachea of animals produce a well-marked diphtheritic exudation with development of the bacilli and secondary involvement of the lymph-glands, in which remarkable necrotic areas occur, with fragmentation of the nuclei (Flexner). Brieger and Fränkel have separated from the cultures a tox-albumin, which, injected into animals, produces paralysis, nephritis, and albuminuria. This point tends strongly to confirm the view that this bacillus is really the infective agent in the disease. It is one of the most virulent poisons known, and when injected in a sufficiently small though fatal dose, there may be no symptoms for days, and the animal may not develop the paralysis for weeks or even months after the injection. A point of very great interest is the fact that cultures from cases vary in virulence, and this is in accord with the remarkable variation in the intensity of different epidemics and different cases. As a rule there is a correspondence between the virulence of the bacilli and the gravity of the case.

Associated with the Klebs-Loeffler bacillus are other pathogenic bacteria, which probably play an important *rôle* in the complications of the disease. Thus streptococci and staphylococci are frequently present in the exudate, and to their invasion through the abraded mucosa are due the secondary suppurations and inflammations of serous surfaces, and to the aspiration of the streptococci into the lungs the common and fatal broncho-pneumonia.

Diphtheria may then be said to be caused by the Klebs-Loeffler bacillus. The production of a false membrane is the local or primary effect; the constitutional symptoms are due to the absorption of the poison in varying doses, while the secondary inflammations are associated with the invasion of the ubiquitous pus organisms.

Pseudo-diphtheritic Processes.—Many substances have the power of exciting pseudo-membranous or croupous inflammation, the exudate of which is not distinguishable from that of diphtheria. Some of them are non-microbic, as steam, ammonia, and chlorine; others are dependent upon micro-organisms, and must be distinguished from true diphtheria.

(*a*) There are cases of pseudo-membranous angina, associated with which is a bacillus identical, morphologically and in its behavior on culture media, with the Klebs-Loeffler bacillus, but which is not pathogenic—i. e., does not produce the tox-albumin, and is harmless when inoculated. Whether this is an attenuated form, as Roux and Yersin hold, is not yet settled. This complicates the question of diagnosis. A patient in my ward presented a thin, grayish pseudo-membrane over the tonsils and fauces, without fever and without constitutional disturbance. Non-pathogenic bacilli, identical with those of true diphtheria, were found by Welch

and Abbott. We need additional information upon the occurrence of this form and its relation to the virulent bacillus.

(*b*) The pseudo-membranous angina of the eruptive fevers is an affection distinct, etiologically at least, from true diphtheria. In a majority of all these cases, particularly in scarlet fever, the Klebs-Loeffler bacillus is absent, and this is in accord with the fact that scarlatinal angina rarely communicates diphtheria, and is still more rarely followed by paralysis.

Streptococci and staphylococci are present in the membranes in these cases. Late in the disease infection with the *bacillus diphtheriæ* may occur, and it is probable that under these circumstances alone is the angina followed by symptoms of paralysis.

Morbid Anatomy.—A majority of the cases die of the faucial or of the laryngeal disease. The exudation may occur in the mouth and cover the inner surfaces of the cheeks; it may even extend beyond the lips on to the skin. This was met once in thirty autopsies at the Montreal General Hospital. The amount of exudation varies in different cases. Usually the tonsils and the pillars of the fauces are swollen and covered with the false membrane. More commonly, in the fatal cases, the exudation is very extensive, involving the uvula, the soft palate, the posterior nares, and the lateral and posterior walls of the pharynx. These parts are covered with a dense pseudo-membrane, in places firmly adherent, in others beginning to separate. In extreme cases the necrosis is advanced and there is a gangrenous condition of the parts. The membrane is of a dirty-greenish or gray color, and the tonsils and palate may be in a state of necrotic sloughing. The erosion may be deep enough in the tonsils to open the carotid artery, or a false aneurism may be produced in the deep tissues of the neck. The nose may be completely blocked by the false membrane, which may also extend into the conjunctivæ and through the Eustachian tubes into the middle ear. In cases of laryngeal diphtheria the exudate in the pharynx may be extensive. In many cases, however, it is slight upon the tonsils and fauces and abundant upon the epiglottis and the larynx, which may be completely occluded by false membrane. In severe cases the exudate extends into the trachea and to the bronchi of the third or fourth dimension. This occurred in nearly half of my thirty Montreal autopsies.

In all these situations the membrane varies very much in consistency, depending greatly upon the stage at which death has occurred. If death has occurred early, it is firm and closely adherent; if late, it is soft, shreddy, and readily detached. When firmly adherent it is torn off with difficulty and leaves an abraded mucosa. In the most extreme cases, in which there is extensive necrosis, the parts look gangrenous. In fatal cases the lymphatic glands of the neck are enlarged and there is a general infiltration of the tissues with serum; the salivary glands, too, may be swollen. In rare instances the membrane extends to the gullet and stomach.

Histological Changes.—We owe largely to the labors of Wagner, Wei-

gert, and more particularly to the splendid work of Oertel, our knowledge of the minute changes which take place in diphtheria. The following is a brief abstract of the views of the last-named author:

The diphtheritic poison induces first a necrosis or death of cells with which it comes in contact, particularly the superficial epithelium and the leucocytes. The deeper cells of the mucosa and of the other parts reached by the poison may also be affected. The second change is hyaline transformation of the dead cells, or, as Weigert terms it, the production of coagulation-necrosis. The bacilli excite inflammation with the migration of leucocytes, which are destroyed by the poison and undergo the hyaline change. The superficial epithelial layers undergo a similar alteration, and what we know as the false membrane represents an aggregation of dead cells, most of which have undergone the transformation into hyaline material. This is in all probability a conservative process by which, in a measure, the poison is localized and prevented from reaching the deeper structures. The laminated condition of the exudate is probably produced by the inflammation of different layers. The formation of these foci of necrobiosis, starting from the epithelium and proceeding inward, is, according to Oertel, the distinguishing characteristic of diphtheria. The action of the poison is by no means confined to the superficial mucosa on which the bacilli grow. Although they do not themselves penetrate deeply, the contiguous bronchial glands show extensive foci of necrosis. In severe cases these necrotic areas are found in the internal organs, in the solitary glands of the intestines, and in the mesenteric glands.

The blood-vessels may themselves be much altered and the capillaries may show extensive hyaline degeneration. Every one of the histological changes described by Oertel in human diphtheria may be paralleled in the experimental disease induced by the Klebs-Loeffler bacillus, particularly the necrotic areas in the deep-seated organs, associated in the lymph-glands with a remarkable fragmentation of the nuclei.

The changes in the *other organs* are variable. When death has occurred from asphyxia there is general congestion of the viscera.

Capillary bronchitis, areas of collapse, and patches of broncho-pneumonia are almost constantly found in fatal cases. In very malignant cases the blood may be fluid. Fibrinous coagula may be found in the heart, but the wide-spread idea that they may cause sudden death is erroneous. Myocardial changes are not infrequent, and in certain cases sudden death is due to heart-failure in consequence of degeneration of the muscle-fibres. Endocarditis is extremely rare. It was not present in one of my thirty autopsies. The serous membranes often show ecchymoses. The kidneys present parenchymatous changes, such as are associated with acute febrile affections. There may, however, be acute nephritis. The spleen and liver show the usual febrile changes. The spleen is, however, not always enlarged.

Symptoms.—The period of incubation varies. In the cases of acci-

dental inoculation the duration is from two to three days. In cases in which the disease is contracted in the usual way it is from seven to twelve days. The initial symptoms are those of an ordinary febrile attack: slight chilliness, fever, and aching pains in the back and limbs. In mild cases these symptoms are trifling, and the child may not feel ill enough to go to bed. Usually the temperature rises to 103° or even more. There may be convulsions at the outset. In an attack of ordinary severity there is at first redness of the fauces, and the child complains of slight difficulty in swallowing. The exudate first appears upon the tonsils. It may be difficult to distinguish the patchy diphtheritic pellicle from the exudate in the tonsillar crypts. The swelling of the throat increases and the glands of the neck become involved. Usually by the third day the membrane has covered the tonsils, and crept on to the pillars of the fauces, and even to the uvula, which is now thickened and œdematous and completely fills the space between the swollen tonsils. The false membrane may extend also to the posterior wall of the pharynx. At first grayish white in color, it changes to a dirty gray, often a yellowish gray. The membrane is firmly adherent, and if removed leaves a bleeding, somewhat eroded surface. New membrane rapidly forms in place of that removed. The general condition of the patient, in a case of moderate severity, is fairly good. The temperature is not necessarily high, and in the absence of complications the range is from 102° to 103°.

At this stage, say the fourth or fifth day of the disease, the condition of the child is favorable. The pulse and temperature are not much above 100°; the throat symptoms are not of extreme severity; and the constitutional depression is not extreme. The symptoms may then abate and the swelling of the neck diminish. The false membrane separates, and by the eighth or tenth day the throat is clear and convalescence begins.

Deviations from this favorable course result either from extension of the local disease or from systemic infection.

(1) **Extension.**—The inflammation may pass into the posterior nares, obstructing the respiration, causing a very acrid and fœtid discharge, and usually a marked aggravation of the constitutional symptoms. The glandular inflammation is usually more intense; due, as Jacobi points out, to the greater richness of the nasal mucosa in lymphatics, which thus favors systemic infection. Though usually secondary, nasal diphtheria may be primary. It greatly increases the danger in any case. From the nose the inflammation may extend through the tear-ducts to the conjunctivæ and into the antra. In these cases the disease is more apt to involve the ears, through the Eustachian tubes, causing otitis media and perforation of the drum.

Extension of the inflammation downward into the larynx is by far the most serious complication of the disease. It is particularly dangerous in children, because it produces what is known as diphtheritic croup. The symptoms are identical with those of ordinary membranous croup.

In many instances the pharynx is but slightly involved. There may be only a trifling patch upon one tonsil. The first symptoms of laryngeal affection are huskiness of the voice, a brazen cough, and stridulous, noisy inspiration and expiration. With increasing obstruction the respiration becomes greatly embarrassed, the lower thoracic zone and the lower sternum are drawn in with each inspiration, and the supra-clavicular and intercostal spaces are depressed. Too often there is a gradually deepening cyanosis, and the child dies asphyxiated.

The exudation may extend into the trachea and bronchi, which become lined by a uniform sheeting of false membrane. It is not always easy to say, during life, whether exudation has taken place into these parts. In the performance of tracheotomy, when membrane is found in the trachea the outlook is generally bad. Occasionally the tracheal and bronchial membrane is coughed up as a definite mould.

(2) **Systemic Infection.**—In mild cases of diphtheria the constitutional disturbance is very slight. There may even be extensive local disease without great constitutional disturbance. As a rule, however, the general symptoms bear a definite proportion to the severity of the local disease. There are rare instances in which from the outset, even before the pharyngeal symptoms are at all well-marked, the constitutional prostration is extreme, the pulse frequent and small, the fever high, the nervous phenomena are pronounced; and the patient sinks in two or three days, overwhelmed by the severity of the poison. In some of these cases the exudation is chiefly nasal; in others the exudation is marked, but the throat symptoms are by no means extensive. It is specially to be noted that the temperature may not be raised; it may even be subnormal. The malignant diphtheria of this kind is fortunately rare. The severe systemic symptoms appear more commonly at a later date, when the pharyngeal symptoms are at their height. They are invariably met when the disease is extensive and when there is a sloughing fœtid condition in the pharynx causing an offensive odor of the breath. The lymphatic glands are greatly enlarged; the pallor is extreme, the color of the face an ashen gray, the pulse is rapid and feeble, and the temperature sinks below normal. In the most aggravated form there are gangrenous processes in the throat. If life is prolonged there may even be extensive sloughing in the tissues of the neck.

There are, of course, many variations in the above clinical picture. The cases may be so mild as scarcely to be recognized. Such cases, indeed, are often mistaken for ordinary lacunar tonsillitis. There are also certain anomalous forms which may be mentioned; cases which come on insidiously, with a tonsillitis of so mild a grade that it may be overlooked, and which is followed by a diphtheritic croup or a severe broncho-pneumonia. In rare instances the disease may almost be termed chronic, since the membrane remains upon the tonsils and pharynx for weeks.

There are instances in which well-characterized pseudo-membrane occurs on the tonsils and fauces without much swelling and without severe constitutional disturbance. A young woman came to my clinic at the University Hospital, Philadelphia, whose tonsils, soft palate and uvula were covered with a smooth, firm, grayish-white pseudo-membrane. There was little or no swelling of the parts, the membrane was clean, its edges were well defined, and on removal of the membrane the mucosa beneath bled freely. The exudation had all the characters of false membrane. The patient had scarcely any constitutional disturbance. The temperature was below 100°, and she had not felt ill enough to go to bed. After persisting for eight or nine days the membrane was gradually removed, and she recovered without any ill effects. The membrane may appear first upon the mucous membrane of the mouth, or it may attack the conjunctiva or the external auditory meatus. Occasionally the vulva, prepuce, or anus is first attacked. In rare cases the skin is involved. When the disease is epidemic, external wounds and abrasions are apt to be infected. In recently delivered women the disease may attack the uterus or vulva.

Complications and Sequelæ.—Local complications, hæmorrhage from the nose or throat, may occur in the severe ulcerative cases. Skin rashes are not infrequent, particularly the diffuse erythema. Occasionally there is urticaria and in the severe cases purpura. The pulmonary complications are extremely important. Fatal cases almost invariably show capillary bronchitis with broncho-pneumonia and large patches of collapse. In very bad cases, with extensive sloughing, the septic particles may reach the bronchi and excite gangrenous processes which may lead to severe and fatal hæmorrhage.

Renal complications are common. In my experience *albuminuria* is present in all severe cases. It may cause with the usual tests only a slight turbidity of the urine, the ordinary febrile albuminuria. In others there is a large amount of albumen, curdy in character. It is only when the albumen is in considerable quantity and associated with epithelial or blood casts that the condition indicates parenchymatous nephritis and is alarming. The nephritis may be quite early in the disease. It sets in occasionally with complete suppression of the urine. In comparison with scarlet fever the renal changes lead less frequently to general dropsy. In the large number of cases of diphtheria which came under observation at the Montreal General Hospital, I call to mind only one or two instances in which the nephritis was associated with general anasarca. Arthritis is an occasional complication just as in scarlet fever. Endocarditis, pericarditis and pleurisy are very rare events.

Of the sequelæ of diphtheria, *paralysis* is by far the most important. This can be experimentally produced in animals, as already noted, by the inoculation of the toxic albumen produced by the bacilli. The paralysis occurs in a variable proportion of the cases, ranging from 10 to 15 and

even to 20 per cent. It is strictly a sequel of the disease, coming on usually in the second or third week of convalescence. Occasionally it comes as early as the seventh or eighth day of the disease. It may follow very mild cases; indeed, the local disease may be so trifling that the onset of the paralysis alone calls attention to the true nature of the trouble.

The disease is a toxic neuritis, due to the absorption of the poison, and, like other forms of multiple neuritis, has an extremely complex symptomatology, according to the nerves which are affected. The paralysis may be local or general.

Of the local paralyses the most common is that which affects the palate. This gives a nasal character to the voice, and, owing to a return of liquids through the nose, causes a difficulty in swallowing. This may be the only symptom. The velum is seen to be relaxed and motionless, and the sensation in it is also much impaired. The affection may extend to the constrictors of the pharynx, and deglutition become embarrassed. Within two or three weeks or even a shorter time the paralysis disappears. In many cases the affection of the palate is only part of a general neuritis. Of other local forms perhaps the most common are paralysis of the eye-muscles, intrinsic and extrinsic. There may be strabismus, ptosis, and loss of power of accommodation. The neuritis may be confined to the nerves of one limb, though more commonly the legs or the arms are affected together. Very often with the palatal paralysis is associated a weakness of the legs without definite palsy but with loss of the knee-jerk.

By far the most important local paralysis is met with in connection with the heart. There may be great retardation, even to thirty beats in the minute. Bradycardia and tachycardia may alternate in the same patient. Heart-failure and fatal syncope may occur at the height of the disease or during convalescence. If they occur during the fever, the child, perhaps after an exaggeration of symptoms, presents an unusual pallor. The pulse becomes weak and rapid, but may fall to fifty, forty, or even lower. The extremities are cold, the temperature sinks, and death takes place, with all the features of collapse, within a few hours. More frequently the fatal collapse comes during convalescence, even as late as the sixth or seventh week after apparent recovery. The attack may set in abruptly, perhaps following a sudden exertion. More commonly there have been symptoms pointing to disturbed cardiac rhythm, or even fainting-spells. In some instances vomiting has preceded the serious cardiac attack. There may be no physical signs other than slight increase in the cardiac dulness and a gallop-rhythm indicating dilatation. These symptoms were formerly ascribed to cardiac thrombosis or to endocarditis. Possibly in some of the cases the result is due, as pointed out by Mosler and Leyden, to an infectious myocarditis, but in a majority of the cases the symptoms are probably due to a neuritis of the cardiac nerves.

The multiple form of diphtheritic neuritis is not uncommon. It may begin with the palatal affection, or with loss of power of accommodation

and loss of the tendon reflexes. This last is an important sign, which, as Buzzard and R. L. MacDonnell have shown, may occur early, but is not necessarily followed by other symptoms of neuritis. There is paraplegia, which may be complete or involve only the extensors of the feet. The disease may extend and involve the arms and face and render the patient entirely helpless. The muscles of respiration may be spared. The chief danger in these severer forms comes from the involvement of the heart and of the muscles of respiration; but the outlook is in many cases not so bad as the patient's condition would indicate. Of thirteen cases collected by Cadet de Gassicourt six died. The sphincters may be involved, though they are often spared.

Diagnosis.—Early in the disease it may be difficult to distinguish diphtheria from follicular tonsillitis. In mild cases it is sometimes impossible. In diphtheria the exudation forms a definite, uniform patch, situated on a deeply congested area of mucosa. In follicular tonsillitis, when the exudate oozes and if the material from the crypts coalesces, it may be extremely difficult to make a diagnosis. If the process is confined to the tonsils, the nature of the case may be dubious. If, however, it extends to the pillars of the fauces and if laryngeal symptoms develop, all doubts are removed. Occasionally the true character of the disease is not manifested until a paralysis develops during convalescence. It is in these cases that the detection of the Klebs-Loeffler bacillus will be of the greatest service in making clear the diagnosis. Cover-glass preparations may be made from the membrane. Cultures should be made in the blood-serum and bouillon mixture, and inoculations performed on animals. Unfortunately, these procedures can scarcely be carried out except in well-equipped laboratories, and a ready and certain clinical method, such as we have for the tubercle bacillus, is not yet available.

Between diphtheritic laryngitis and croup a majority of writers now hold that there is no essential difference; but it is more rational to believe that there is a non-specific pseudo-membranous laryngitis. This is a point, too, which bacteriology may be able to clear up. In several cases which have been examined the Loeffler bacillus has been present. The diagnosis between the two conditions is by no means easy. In the diphtheritic form, however, there is almost invariably exudation upon the tonsils or soft palate. Between scarlet fever and diphtheria there may be some confusion The question has already been discussed.

The recognition of the diphtheritic paralysis offers no difficulties.

Prognosis.—In hospital practice the disease is very fatal, owing largely to the fact that only the severer forms are admitted. In country places epidemics may display an appalling virulence and kill nearly all the children attacked. In cases of ordinary severity the outlook is usually good. Death results from involvement of the larynx, septic infection, sudden heart-failure, diphtheritic paralysis, occasionally from uræmia, and sometimes from broncho-pneumonia developing in the convalescence.

Treatment.—Prophylaxis.—Cases of diphtheria should invariably be isolated. Physicians should insist that other children in the family be kept from school and from mingling with their schoolmates. All clothing and utensils which have been used by the patient should be thoroughly disinfected. For this purpose the clothing may be soaked for twenty-four hours and then boiled in a two-per-cent carbolic solution. For disinfecting the room sulphur fumigation may be employed, taking care that the air is rendered moist, or the floor and walls should be thoroughly scoured with corrosive-sublimate solution.

Caillé has urged the importance of a careful inspection of the tonsils and mouth in children, special attention being paid to the care of the teeth and to the tonsils, which, if swollen and irregular, should be removed. In persons liable to exposure Loeffler recommends the use of antiseptic mouth-washes, such as sublimate (1 to 10,000), chlorine-water (1 to 1,100), or thymol. After recovery at least two weeks should elapse before the child is permitted to mingle with others or to return to school.

Recently it has been announced that the blood-serum of animals rendered secure against the diphtheritic bacillus and its products can nullify the effects of the poison of diphtheria.

General Treatment.—The two indications in the treatment of diphtheria are to prevent or limit the local development of the bacilli and to combat the effects of the toxic materials which they produce.

The usual measures should be employed to insure thorough cleanliness and ventilation and to diminish the danger of infection. The air should be kept moist with steam. Mild cases require but little treatment. A fair quantity daily of liquid food, with ice to suck, and a gargle of chlorate of potash are sufficient. In more severe cases the greatest care should be taken to maintain the strength of the patient. The food should be given at stated intervals. Stimulants will be required early and should be given freely. In very young children with the pharyngeal involvement swallowing is painful, and the giving of food by the mother or nurse is a continuous struggle. In such instances nutritive enemata should be used.

We are still without a remedy capable of combating in any way the effects of the poisonous tox-albumins. Two remedies are warmly recommended—the tincture of the perchloride of iron, which may be given hourly in four or five drop doses to a child of three, and the corrosive sublimate, of which a child a year old may take as much as half a grain a day. Personally, I much prefer the perchloride of iron; and I cannot say that I have seen from the mercury, given either as the bichloride or as calomel, the specially good effects which many writers describe. I have not seen any good follow the administration of the sulphides or the benzoates or quinine in large doses. Peroxide of hydrogen has been warmly recommended.

Local Treatment.—Diphtheria is a local disease at first, and by the production of poisonous substances causes the severe systemic symptoms.

Hence the importance of local treatment. It is not well to attempt forcibly to remove the false membranes, though some writers recommend that they should be scraped off. As far as possible thorough cleanliness and disinfection of the fauces should be insured by repeatedly spraying, either with carbolic acid, corrosive sublimate (two grains to the pint), chlorine-water, boric acid, Condy's fluid, salicylic acid or peroxide of hydrogen (50 per cent solution), or local application of sulphur with iodoform is recommended. The tonsils and fauces may be thoroughly swabbed every hour or two with a solution of carbolic acid (℥xv) and perchloride of iron (℥ ijss.) in glycerin (℥ j) and water (℥ j). Agents which are believed to dissolve the membrane are lactic acid, which may be employed with lime-water (two drachms to six ounces) and trypsin (thirty grains to the ounce).

Pepsin has also been used, and the vegetable pepsin, which may be mixed with water and glycerin.

Nasal diphtheria requires prompt and thorough disinfection of the passages. The best solutions are those recommended by Jacobi—chloride of sodium, saturated boric acid, or one part of bichloride of mercury, thirty-five of chloride of sodium, and one thousand of water, or the one per cent solution of carbolic acid. The solution may be applied with a syringe or a spray. To be effectual the injection must be properly given. The nurse should be instructed to pass the nozzle of the syringe horizontally, not vertically; otherwise the fluid will return through the same nostril. In refractory children there is sometimes great difficulty in giving these injections, in which case suppositories of boric acid may be employed, but they are not efficient substitutes.

When the larynx becomes involved a steam tent may be arranged upon the bed, so that the child may breathe an atmosphere saturated with moisture. If the dyspnœa becomes urgent, an emetic of sulphide of zinc or ipecacuanha may be given. When the signs of obstruction are marked, however, there should be no delay in the performance of intubation or tracheotomy. The diphtheritic paralysis requires rest in bed, and the avoidance of sudden exertion, particularly in those cases in which the heart-rhythm is disturbed. In the chronic forms with wasting, massage, electricity and strychnine are invaluable aids. If swallowing becomes very difficult, the patient must be fed with the stomach-tube, which is very much preferable to feeding per rectum.

XVI. ERYSIPELAS.

Definition.—An acute, contagious disease, characterized by a special inflammation of the skin caused by streptococci.

Etiology.—Erysipelas is a wide-spread affection, endemic in most communities, and at certain seasons epidemic. We are as yet ignorant of

the atmospheric or telluric influences which favor the diffusion of the poison.

It is particularly prevalent in the spring of the year. This was very noticeable in the Philadelphia Hospital, in which the erysipelas wards were usually empty except in the spring and autumn months. The affection prevails extensively in old ill-ventilated hospitals and institutions in which the sanitary conditions are defective. With the improved sanitation of late years the number of cases has materially diminished. It has been observed, however, to break out in new institutions under the most favorable hygienic circumstances. Erysipelas is both contagious and inoculable; but, except under special conditions, the poison is not very virulent and does not seem to act at any great distance. It can be conveyed by a third person. The poison certainly attaches itself to the furniture, bedding, and walls of rooms in which patients have been confined.

The disposition to the disease is wide spread, but the susceptibility is specially marked in the case of individuals with wounds or abrasions of any sort. Recently delivered women and persons who have been the subject of surgical operations are particularly prone to it. A wound, however, is not necessary, and in the so-called idiopathic form, although it may be difficult to say that there was not a slight abrasion about the nose or lips, in very many cases there certainly is no observable external lesion.

Chronic alcoholism, debility, and Bright's disease are predisposing agents. Certain persons show a special susceptibility to the disease, and it may recur in them repeatedly. There are instances, too, of a family predisposition to the disease.

The specific agent of the disease appears to be a streptococcus which has been very thoroughly studied by Koch and Fehleisen. It was believed at first to have specific and peculiar morphological properties, but it is now generally held that it cannot be distinguished by any biological or chemical tests from the *streptococcus pyogenes*.

Morbid Anatomy.—Erysipelas is a simple inflammation. In its uncomplicated forms there is seen, post mortem, little else than inflammatory œdema. Investigations have shown that the cocci are found chiefly in the lymph-spaces and most abundantly in the zone of spreading inflammation. In the uninvolved tissue beyond the inflamed margin the micrococci are to be found in the lymph-vessels, and it is here, according to Metschnikoff and others, that an active warfare goes on between the leucocytes and the cocci (phagocytosis). In more extensive and virulent forms of the disease there is usually suppuration. It is stated that the inflammation may pass inward from the scalp through the skull to the meninges. This I have never seen, but in one case I traced the extension from the face along the fifth nerve to the meninges, where an acute meningitis and thrombosis of the lateral sinus were excited.

The visceral complications of erysipelas are numerous and important. The majority of them are of a septic nature. Infarcts occur in the lungs,

spleen, and kidneys, and there may be the general evidences of pyæmic infection.

Some of the worst cases of malignant endocarditis are secondary to erysipelas; thus of twenty-three cases three occurred in connection with this disease. Septic pericarditis and pleuritis also occur.

As just mentioned, the disease may in rare cases extend and involve the meninges. Pneumonia is not a very common complication.

Acute nephritis is also met with. It is often ingrafted upon an old chronic trouble.

Symptoms.—The following description applies specially to erysipelas of the face and head, the form of the disease which the physician is most commonly called upon to treat.

The *incubation* is variable, probably from three to seven days.

The stage of *invasion* is often marked by a rigor, and followed by a rapid rise in the temperature and all the characters of an acute fever. When there is a local abrasion, the spot is slightly reddened; but if it is idiopathic, there is seen within a few hours slight redness over the bridge of the nose and on the cheeks. The swelling and tension of the skin increase and within twenty-four hours the external symptoms are well marked. The skin is smooth, tense, and œdematous. It looks red, feels hot, and the superficial layers of the epidermis may be lifted as small blebs. The patient complains of an unpleasant feeling of tension in the skin; the swelling rapidly increases; and during the second day the eyes are usually closed. The first-affected parts gradually become pale and less swollen as the disease extends at the periphery. When it reaches the forehead it progresses as an advancing ridge, perfectly well defined and raised; and often, on palpation, hardened extensions can be felt beneath the skin which is not yet reddened. Even in a case of moderate severity, the face is enormously swollen, the eyes are closed, the lids greatly œdematous, the ears thickened, the scalp is swollen, and the patient's features are quite unrecognizable. The formation of blebs is common on the eyelids, ears, and forehead. The cervical lymph-glands are swollen, but are usually masked in the œdema of the neck. The temperature keeps high without marked remissions for four or five days and then defervescence takes place by crisis. The general condition of the patient varies much with his previous condition of health. In old and debilitated persons, particularly in those addicted to alcohol, the constitutional depression from the outset may be very great. Delirium is present, the tongue becomes dry, the pulse feeble, and there is marked tendency to death from toxæmia. In the majority of cases, however, even with extensive disease, the constitutional disturbance, considering the height of the fever range, is slight. The mucous membrane of the mouth and throat may be swollen and reddened. The erysipelatous inflammation may extend to the larynx, but the severe œdema of this part occasionally met with is commonly due to extension of the inflammation from without inward.

There are cases in which the inflammation extends from the face to the neck, and over the chest, and may gradually migrate or wander over the greater part of the body (*E. migrans*).

The close relation between the erysipelas coccus and the pus organisms is shown by the frequency with which suppuration occurs in facial erysipelas. Small cutaneous abscesses are common about the cheeks and forehead and neck, and beneath the scalp large collections of pus may accumulate. Suppuration seems to occur more frequently in some epidemics than in others, and at the Philadelphia Hospital one year nearly all the cases in the erysipelas wards presented local abscesses.

Complications.—Meningitis is rare. The cases in which death occurs with marked brain symptoms do not usually show, post mortem, meningeal affection. The delirium and coma are due to the fever, or to toxæmia.

Pneumonia is an occasional complication. Ulcerative endocarditis and septicæmia are more common. Albuminuria is almost constant, particularly in persons over fifty. True nephritis is occasionally seen. Da Costa has called attention to curious irregular returns of the fever which occur during convalescence without any aggravation of the local condition.

The diagnosis rarely presents any difficulty. The mode of onset, the rapid rise in fever, and the characters of the local disease are quite distinctive. Acute necrosis of bone may sometimes be regarded as erysipelas, a mistake which I once saw made in connection with the lower end of the femur.

Prognosis.—Healthy adults rarely die. In the new-born, when the disease attacks the navel, it is almost always fatal. This is probably an acute septic infection. In alcoholic subjects and in the aged erysipelas is a serious affection, and death may result either from the intensity of the fever or, more commonly, from toxæmia. The wandering or ambulatory erysipelas, which has a more protracted course, may cause death from exhaustion.

Treatment.—Isolation should be strictly carried out, particularly in hospitals. A practitioner in attendance upon a case of erysipelas should not attend cases of confinement.

The disease is self-limited and a large majority of the cases get well without any internal medication. I can speak definitely on this point, having, at the Philadelphia Hospital, treated many cases in this way. The diet should be nutritious and light. Stimulants are not required except in the old and feeble. For the restlessness, delirium, and insomnia, chloral or the bromides may be given; or, if these fail, opium. When fever is high the patient may be bathed or sponged, or, in private practice, if there is an objection to this, antipyrin or antifebrin may be given.

Of internal remedies believed to influence the disease, the tincture of the perchloride of iron has been highly recommended. At the Montreal

General Hospital this was the routine treatment, and doses of half a drachm to a drachm were given every three or four hours. I am by no means convinced that it has any special action; nor, so far as I know, has any medicine, given internally, a definite control over the course of the disease.

Of local treatment, the injection of antiseptic solutions at the margin of the spreading areas has been much practised. Two per cent solutions of carbolic acid, the corrosive sublimate and the biniodide of mercury have been much used. The injection should be made not into but just a little beyond the border of the inflamed patch. F. P. Henry has treated a large number of cases at the Philadelphia Hospital with the latter drug, and this mode of practice is certainly most rational.

Of local applications, ichthyol is at present much used. The inflamed region may be covered with salicylate of starch. Perhaps as good an application as any is cold water, which was highly recommended by Hippocrates.

XVII. SEPTICÆMIA AND PYÆMIA.

1. SEPTICÆMIA.

Definition.—A general febrile infection, without foci of suppuration, which results from the absorption of toxic materials produced by bacteria. The organisms producing septicæmia are, as a rule, those of suppuration—namely, the forms of streptococci and staphylococci.

Clinical Forms.*—(*a*) *Fermentation Fever.*—This is also known as the resorption fever, aseptic fever, or after fever, and is the simplest of all wound complications. It is the febrile process which is produced after transfusion or the injection of pepsin into the blood. The term fermentation fever was employed by Bergman, as he held that it was caused by the absorption of the fibrin ferments. This fever may follow an injury or operation, particularly if there has been necrosis of the superficial tissues by the solutions used in the dressing. It may also follow the extravasation of blood, particularly when under pressure or tension.

The fever, which appears a few hours after the injury or operation, is not preceded by a chill. It usually reaches its height rapidly, sometimes rising to 103° or 104°. The constitutional disturbance is not great, and it subsides spontaneously in from one to three days. This form is ranked as a septicæmia, since the ferment acts in a manner similar to the toxins produced by micro-organisms. It is not yet certain that bacteria do not play an important part in its production.

(*b*) *Sapræmia.*—This is a septic intoxication caused by the ptomaines produced in wounds by the putrefactive bacteria. There are various forms of these organisms; some are bacilli, others belong to the proteus group.

* I follow here the division in Senn's Principles of Surgery.

In their growth, chemical poisons (toxins) are produced, and under the term sapræmia is included the group of symptoms caused by the absorption of these toxins from any local focus of putrefaction.

The symptoms vary with the dose absorbed. Twenty-four hours, or later, after the injury or operation a chill initiates the constitutional disturbance; the fever rises rapidly, reaching 103° or 104°; the pulse is quick, and there may, in severe cases, be great prostration. Nervous symptoms are common—headache, restlessness, and delirium. The tongue is dry, often glazed, and there may at first be gastric irritation. The clinical picture is that of a severe infection. Three conditions must be present in this form of sepsis—dead tissue, infection of this dead tissue with putrefactive bacteria, and a sufficient time to have enabled the putrefactive bacteria to produce a toxic quantity of ptomaines (Senn). The necrotic tissue may be the blood-clot in a wound, the tissues in the interior of the uterus after parturition, or tissues bruised and rendered necrotic by injury or by the action of cold, heat, or chemical substances.

The outlook in sapræmia depends much upon the dose of the poison which has been absorbed and the possibility of removing and cleansing the infected focus.

(*c*) *Progressive Septicæmia.*—In this the septic intoxication is not the result of the bacteria of putrefaction, but organisms enter the blood from some local septic focus. "The intoxication in this form of sepsis is not only caused by ptomaines which are produced at the primary seat of infection, but ptomaines are also produced in the blood by the microbes which it contains" (Senn). The pus microbes are the most frequent cause of this form of septicæmia, and reach the blood either through the wall of the blood-vessels or through the lymph-channels.

The clinical features of this form are well seen in the cases of puerperal septicæmia or in dissection wounds, in which the course of the infection may be traced along the lymphatics. The symptoms usually set in within twenty-four hours, and rarely later than the third or fourth day. There is a chill or chilliness, with moderate fever at first, which gradually rises and is marked by daily remissions and even intermissions. The pulse is small and compressible, and may reach 120 or higher. Gastro-intestinal disturbances are common, the tongue is red at the margin, and the dorsum is dry and dark. There may be early delirium or marked mental prostration and apathy. As the disease progresses there may be pallor of the face or a yellowish tint. Capillary hæmorrhages are not uncommon.

The outlook is always serious. In severe cases death may occur within twenty-four hours, and in fatal cases life is rarely prolonged for more than seven or eight days. On post-mortem examination there may be no focal lesions in the viscera, and the seat of infection may present only slight changes. The spleen is enlarged and soft, the blood may be extremely dark in color, and hæmorrhages are common, particularly on the serous surfaces. Neither thrombi nor emboli are found.

2. PYÆMIA.

Definition.—A general disease, characterized by recurring chills and intermittent fever and the formation of abscesses in various parts, all of which result from the contamination of the blood by products arising from a focus contaminated by the bacteria of suppuration.

Etiology.—As a rule, the disease follows extension of suppuration about a wound or the collection of pus in some part. It was thought at first that the pus itself was taken up by the blood. Virchow showed the important part played by thrombosis and embolism. The works of Lister, Klebs, Pasteur, Koch, and others have demonstrated the important *rôle* of micro-organisms in the disease. The pus microbes are the *streptococcus pyogenes* and forms of *staphylococci*. The streptococcus is most frequently found in the pus at the primary seat and in the metastatic abscesses.

The process which takes place is as follows: In a suppurating wound, for example, the pus organisms induce coagulation-necrosis in the smaller vessels with the production of thrombi and purulent phlebitis. The entrance of pus organisms in small numbers into the blood does not necessarily produce pyæmia. Commonly the transmission to various parts from the local focus takes place by the fragments of thrombi which pass as emboli to different parts, where if the conditions are favorable the pus organisms excite suppuration. A thrombus which is not septic or contaminated, when dislodged and impacted in a distant vessel, produces only a simple infarction; but, coming from an infected source and containing pus microbes, an independent centre of infection is established wherever the embolus may lodge. These independent suppurative centres in pyæmia, known as *embolic* or *metastatic abscesses*, have the following distribution:

(*a*) In external wounds, in osteo-myelitis, and in acute phlegmon of the skin, the embolic particles very frequently excite suppuration in the lungs, producing the well-known wedge-shaped pyæmic infarcts; but in some cases the infected particles pass through the lungs, and there are foci of inflammation in the heart and kidneys.

(*b*) Suppurative foci in the territory of the portal system, particularly in the intestines, produce metastatic abscesses in the liver with or without suppurative pyle-phlebitis.

(*c*) An interesting form of medical pyæmia is produced by malignant endocarditis—the arterial pyæmia of Wilks—in which, as a result of inflammation of the endocardium (either secondary to suppurative disease elsewhere, or following the infection of pneumonia or of certain general diseases), showers of infected thrombi are conveyed from the vegetations in the left heart and produce multiple abscesses in the spleen, kidneys, intestines, brain, and even in the skin.

(*d*) There are cases of so-called *idiopathic pyæmia* in which the pri-

mary focus of the disease is not apparent, but in which there are multiple abscesses in various parts of the body.

Symptoms.—In a case of wound infection, prior to the onset of the characteristic symptoms, there may be signs of local trouble, and, if a discharging wound, the pus may change in character. The onset of the disease is marked by a severe rigor, during which the temperature rises to 103° or 104° and is followed by a profuse sweat. These chills are repeated at intervals, either daily or every other day. In the intervals there may be slight pyrexia. The constitutional disturbance is marked and there are loss of appetite, nausea, and vomiting, and, as the disease progresses, rapid loss of flesh. Transient erythema is not uncommon. Local symptoms usually develop. If the lungs become involved there are dyspnœa and cough. The physical signs may be slight. Involvement of the pleura and pericardium is common. The tint of the skin is changed; at first pale and white, it subsequently becomes bile-tinged. The spleen is enlarged, and there may be intense pain in the side, pointing to perisplenitis from embolism. Usually in the rapid cases a typhoid state is gradually developed, and the patient dies comatose.

In the chronic cases the disease may be prolonged for months; the chills recur at long intervals, the temperature is irregular, and the condition of the patient varies from month to month. The course is usually slow and progressively downward.

Diagnosis.—Pyæmia is a disease frequently overlooked and often mistaken for other affections.

Cases following a wound, an operation, or parturition are readily recognized. On the other hand, the following conditions may be overlooked:

Osteo-myelitis.—Here the lesion may be limited, the constitutional symptoms severe, and the course of the disease very rapid. I recall two instances in which the actual cause of the trouble was discovered only at the post-mortem.

So, too, acute septic infection may follow *gonorrhœa* or a *prostatic abscess*.

Cases are sometimes confounded with *typhoid fever*, particularly the more chronic instances, in which there are diarrhœa, great prostration, delirium, and irregular fever. The spleen, too, may be enlarged.

In some of the instances of *ulcerative endocarditis* the diagnosis is very difficult, particularly in what is known as the typhoid type of this disease, in contradistinction to the septic. In *acute miliary tuberculosis* the symptoms occasionally resemble those of septicæmia, more commonly those of typhoid fever.

The *post-febrile arthritides*, such as occur after scarlet fever and gonorrhœa, are really instances of mild septic infection. The joints may sometimes proceed to suppuration and pyæmia develop. So, also, in *tuberculosis of the kidneys* and *calculous pyelitis* recurring rigors and sweats

due to septic infection are common In this latitude septic and pyæmic processes are too often confounded with *malaria.* In early tuberculosis, or even when signs of excavation are present in the lungs, and in cases of suppuration in various parts, particularly empyema and abscess of the liver, the diagnosis of malaria is made. The practitioner may take it as a safe rule, to which he will find very few exceptions, *that an intermittent fever which resists quinine is not malaria.*

Other conditions associated with chills which may be mistaken for pyæmia are profound anæmia, certain cases of Hodgkin's disease, the hepatic intermittent fever associated with the lodgment of gall-stone at the orifice of the common duct, rare cases of essential fever in nervous women, and the intermittent fever sometimes seen in rapidly developing cancer.

On two or three occasions I have met with intermittent pyrexia persisting for weeks, in which it seemed to be impossible to give any explanation of the phenomena—cases in which tuberculosis, malaria, or septicæmia could be almost positively ex t.

Treatment.—The treatment s septicæmia and pyæmia is largely a surgical problem. The cases which come under the notice of the physician usually have visceral abscesses or ulcerative endocarditis, conditions which are irremediable. We have no remedy which controls the fever. Quinine and the new antipyretics may be tried, but they are of little service. Quinine is probably better than antipyrin and antifebrin, which lower the temperature for a time, but when a careful two-hourly twenty-four-hour chart is taken, it is often found that the depression under the influence of the drug is made up at some other period of the day; a morning may be substituted for an afternoon fever.

The brilliant and remarkable results which follow complete evacuation of the pus with thorough drainage give the indication for the only successful treatment of this condition.

Unfortunately in too many cases which the physician is called upon to treat, the region of suppuration is not accessible, and we have to be content with the employment of general measures for the support of the patient's strength.

XVIII. CHOLERA ASIATICA.

Definition.—A specific, infectious disease, caused by the comma bacillus of Koch, and characterized clinically by violent purging and rapid collapse.

Historical Summary.—Cholera has been endemic in India from a remote period, but only within the present century has it made inroads into Europe and America. An extensive epidemic occurred in 1832, in which year it was brought in immigrant ships from Great Britain to Quebec. It

travelled along the lines of traffic up the Great Lakes; and finally reached as far west as the military posts of the upper Mississippi. In the same year it entered the United States by way of New York. There were recurrences of the disease in 1835–'36. In 1848 it entered the country through New Orleans, and spread widely up the Mississippi Valley and across the continent to California. In 1849 it again appeared. In 1854 it was introduced by immigrant ships into New York and prevailed widely throughout the country. In 1866 and in 1867 there were less serious epidemics. In 1873 it again appeared in the United States, but did not prevail widely. In 1884 there was an outbreak in Europe. Although occasional cases have been brought by ship to the quarantine stations in this country, the disease has not gained a foothold here since 1873.

Etiology.—In 1884 Koch announced the discovery of the specific organism of this disease. Subsequent observations have confirmed his statement that the comma bacillus, as it is termed, occurs constantly in the true cholera, and in no other disease. It has the form of a slightly bent rod, which is thicker, but not more than about half the length of the tubercle bacillus, and sometimes occurs in an S-form. It is not a true bacillus, but really a spirochæte. The organism grows upon a great variety of media and displays distinctive and characteristic appearances. The bacilli are found in the intestine, in the stools from the earliest period of the disease, and very abundantly in the characteristic rice-water evacuations, in which they may be seen as an almost pure culture. They very rarely occur in the vomit. Post mortem, they are found in enormous numbers in the intestine. In acutely fatal cases they do not seem to invade the intestinal wall, but in cases with a more protracted course they are found in the follicles and even in the deeper tissues.

Modes of Infection.

(*a*) **Contagion.**—It appears probable that cholera is not highly contagious in the same sense as small-pox and scarlet fever, but in this respect is very similar to typhoid fever. Physicians, nurses, and others in close contact with the patients are not often affected. On the other hand, such persons as washer women, who are brought into very close contact with the cholera stools and the linen of the cholera patients, are particularly prone to the disease.

(*b*) **Infection.**—The leading authorities now agree that the disease is propagated chiefly by the contamination of water used for drinking, washing, and cooking. It is quite possible that articles of food may be contaminated, particularly vegetables, such as lettuces and cresses and others, which have been washed in infected water; but this is probably a minor danger in comparison with impure drinking-water. The bacilli, under suitable circumstances—that is, when much impurity is present—may develop to some extent in the water; Koch, as is well known, found the bacilli in a tank in India, from which the inhabitants were supplied with water for drinking and washing. Strongly in favor of this view is the fact

that the virulence of an epidemic in any region is generally in direct proportion to the imperfection of the water-supply. On the other hand, with improvements and perfection in the water-works of a place, the epidemics are reduced in intensity, and the place may even obtain immunity against the disease. Not only in India has the demonstration of the connection between drinking-water and cholera infection been amply furnished, but in England there have been many valuable illustrations. One of the most notable of these was the celebrated Broad Street pump, in London, which in 1854 was connected with a severe epidemic. Milk also may possibly in some instances convey the poison.

Pettenkofer, on the other hand, denies the truth of this drinking-water theory, and maintains that the conditions of the soil are of the greatest importance; particularly a certain porosity, combined with moisture and contamination with organic matter, such as sewage. According to him, the condition most favorable for the development of the virus is found when the subsoil water is lowest. As Stillé remarks: "It is more descriptive of the fact to say that so far as cholera has in any way to do with the condition of the soil, it is most apt to be severe and prevalent when very dry weather follows a very wet period. Such instances are most favorable to putrefactive fermentation and the dissemination of its products, which thus reach wells of drinking-water and even rivers, especially when sewers empty into the water."

Pettenkofer holds that germs develop in the subsoil moisture during the warm months, and that they rise into the atmosphere as a miasm.

The disease is always spread along the lines of human travel. In India it has, in many notable cases, been widely spread by pilgrims. It is carried also by caravans and in ships. It is not conveyed through the atmosphere.

Places situated at the sea-level are more prone to the disease than those situated inland. In high altitudes the disease does not prevail so extensively. A high temperature favors the development of the disease, but in Europe and America the epidemics have been chiefly in the late summer and in the autumn.

The disease attacks persons of all ages. It is particularly prone to attack the intemperate and those debilitated by want of food and bad surroundings. Depressing emotions, such as fear, undoubtedly have a marked influence. It is doubtful whether an attack furnishes immunity against a second one.

Morbid Anatomy.—There are no characteristic anatomical changes in cholera; but a post-mortem diagnosis of the nature of the disease could be made by any competent bacteriologist, as the micro-organisms are specific and distinctive. The body has the appearances associated with profound collapse. There is often marked post-mortem elevation of temperature. The *rigor mortis* sets in early and may produce displacement of the limbs. The lower jaw has been seen to move and the eyes to

rotate. Various movements of the arms and legs are also seen. The blood is thick and dark, and there is a remarkable diminution in the amount of water and salts. The peritonæum is sticky, and the coils of intestines are congested and look thin and shrunken. There is nothing special in the appearance of the stomach. The small intestine usually contains a turbid serum, similar in appearance to that which was passed in the stools. The mucosa is, as a rule, pale and swollen and often congested about the Peyer's patches. Post mortem the epithelial lining is sometimes denuded, but this is probably not a change which takes place during life. In the stools, however, large numbers of columnar epithelial cells have been described by many observers. The bacilli are found in the contents of the intestine and in the mucous membrane. The spleen is usually small. The liver and kidneys may show cloudy swelling. The heart is flabby; the right chambers are distended with blood and the left chambers are usually empty. The lungs are collapsed, and congested at the bases.

The above appearances are those met with in cases which prove rapidly fatal. When the patient survives and death occurs during reaction, there may be more definite inflammatory appearances in the intestines and more definite changes in the kidneys and liver.

Symptoms.—A period of incubation of uncertain length, probably not more than from two to five days, precedes the development of the symptoms.

Three stages may be recognized in the attack: the preliminary diarrhœa, the collapse stage, and the period of reaction.

(*a*) *The preliminary diarrhœa* may set in abruptly without any previous indications. More commonly there are, for one or two days, colicky pains in the abdomen, with looseness of the bowels, perhaps vomiting, with headache and depression of spirits. There may be no fever.

(*b*) *Collapse Stage.*—The diarrhœa increases, or, without any of the preliminary symptoms, sets in with the greatest intensity; and profuse liquid evacuations succeed each other rapidly. There are in some instances griping pains and tenesmus. More commonly there is a sense of exhaustion and collapse. The thirst becomes extreme, the tongue is white; cramps of great severity occur in the legs and feet. Within a few hours vomiting sets in and becomes incessant. The patient rapidly sinks into a condition of collapse, the features are shrunken, the skin of an ashy gray hue, the eyeballs sink in the sockets, the nose is pinched, the cheeks are hollow, the voice becomes husky, the extremities are cyanosed, and the skin is shrivelled, wrinkled, and covered with a clammy perspiration. The temperature sinks. In the axilla or in the mouth it may be from five to ten degrees below normal, but in the rectum and in the internal parts it may be 103° or 104°. The pulse becomes extremely feeble and flickering, and the patient gradually passes into a condition of coma, though consciousness is often retained until near the end.

The fæces are at first yellowish in color, from the bile pigment, but soon they become grayish white and look like turbid whey or rice-water; whence the term "rice-water stools." There are found in it numerous small flakes of mucus and granular matter, and at times blood. The reaction is usually alkaline. The fluid contains albumen and the chief mineral ingredient is chloride of sodium. Microscopically, mucus and epithelial cells and innumerable bacteria are seen, the majority of the latter being the comma bacilli.

The condition of the patient is largely the result of the concentration of the blood consequent upon the loss of serum in the stools. There is almost complete arrest of secretion, particularly of the saliva and the urine. On the other hand, the sweat-glands increase in activity, and in nursing women it has been stated that the lacteal flow is unaffected. This stage may not last more than two or three hours, but more commonly lasts from twelve to twenty-four. There are instances in which the patient dies before purging begins—the so-called *cholera sicca.*

(*c*) *Reaction Stage.*—When the patient survives the collapse, the cyanosis gradually disappears, the warmth returns to the skin, which may have for a time a mottled color or present a definite erythematous rash. The heart's action becomes stronger, the urine increases in quantity, the irritability of the stomach disappears, the stools are at longer intervals, and there is no abdominal pain. In the reaction the temperature may not rise above normal. Not infrequently this favorable reaction is interrupted by a recurrence of severe diarrhœa and the patient is carried off in a relapse. Other cases pass into the condition of what has been called *cholera-typhoid*, a state in which the patient is delirious, the pulse rapid and feeble, and the tongue dry. Death finally occurs with coma. These symptoms have been attributed to uræmia.

During epidemics attacks are found of all grades of severity. There are cases of diarrhœa with griping pains, liquid, copious stools, vomiting, and cramps, with slight collapse. The term *cholerine* has been applied to these cases. They resemble the milder cases of *cholera nostras.* At the opposite end of the series there are the instances of *cholera sicca*, in which death may occur in a few hours after the attack, without diarrhœa. There are cases also in which the patients are overwhelmed with the poison and die comatose, without the preliminary stage of collapse.

Complications and Sequelæ.—The typhoid condition has already been referred to. The consecutive nephritis rarely induces dropsy. Diphtheritic colitis has been described. There is a special tendency to diphtheritic inflammation of the mucous membranes, particularly of the throat and genitals. Pneumonia and pleurisy may develop, and destructive abscesses may occur in different parts. Suppurative parotitis is not very uncommon. In rare instances local gangrene may develop. A troublesome symptom of convalescence may be cramps in the muscles of the arms and legs.

Diagnosis.—The only affection with which Asiatic cholera could be confounded is the *cholera nostras*, the severe choleraic diarrhœa which occurs during the summer months in temperate climates. The clinical picture of the two affections is identical. The extreme collapse, vomiting, and rice-water stools, the cramps, the cyanosed appearance, are all seen in the worst forms of cholera nostras. In enfeebled persons death may occur within twelve hours. It is of course extremely important to be able to diagnose between the two affections. This can only be done by one thoroughly versed in bacteriological methods, and conversant with the diversified flora of the intestines. The comma bacillus is present in the dejections of a great majority of the cases and can be seen on cover-glass preparations. Though the eye of the expert may be able to differentiate between the bacillus of true cholera and that which occurs in cholera nostras, cultures should be made, from which alone positive results can be obtained.

Attacks very similar to Asiatic cholera are produced in poisoning by arsenic, corrosive sublimate, and certain fungi; but a difficulty in diagnosis could scarcely arise.

The *prognosis* is always uncertain, as the mortality ranges in different epidemics from 30 to 80 per cent. Intemperance, debility, and old age are unfavorable conditions. The more rapidly the collapse sets in, the greater is the danger. Cases with marked cyanosis and very low temperature rarely recover.

Prophylaxis.—Preventive measures are all-important, and isolation of the sick and thorough disinfection have effectually prevented the disease entering England or the United States since 1873. On several occasions since that date, cholera has been brought to various ports in America, but has been checked at quarantine. During epidemics the greatest care should be exercised in the disinfection of the stools and linen of the patients. When an epidemic prevails, persons should be warned not to drink water unless previously boiled. Errors in diet should be avoided. As the disease is not more contagious than typhoid fever, the chance of a person passing safely through an epidemic depends very much upon how far he is able to carry out thoroughly prophylactic measures. Digestive disturbances are to be treated promptly, and particularly the diarrhœa, which so often is a preliminary symptom. For this opium and acetate of lead and large doses of bismuth should be given.

Attempts have been made to procure a protective virus. During the last epidemic in Europe, Ferran, in Spain, made a large number of inoculations which were claimed to be protective; but the French commission reported adversely against these claims. Shakespeare* seemed to have been more favorably impressed. Both Gamaleia and Lowenthal have ren-

* Report on Cholera in Europe and India. By E. O. Shakespeare, United States Commissioner. Washington, 1891.

dered animals immune against the cholera virus, but it is not probable that the method which they employ would be available for man.

Medicinal Treatment.—During the initial stage, when the diarrhœa is not excessive but the abdominal pain is marked, opium is the most efficient remedy, and it should be given hypodermically as morphia. It is advisable to give at once a full dose, which may be repeated on the return of the pain. It is best not to attempt to give remedies by the mouth, as they disturb the stomach. Ice should be given, and brandy or hot coffee. In the collapse stage, writers speak strongly against the use of opium. Undoubtedly it must be given with caution, but, judging from its effects in cholera nostras, I should say that collapse *per se* was not a contra-indication. For the intense thirst the patient may be given ice-water, of which he should be allowed to drink freely. The vomiting is very difficult to check. In severe cases creosote, hydrocyanic acid, and other remedies seem quite ineffectual.

Salol has been warmly recommended as capable of preventing the development of the bacilli in the intestine.

External applications of heat should be made and a hot bath may be tried. Warm applications to the abdomen are very grateful. Hypodermic injections of ether will be found serviceable.

Judging from the success which has followed the copious enemata in *cholera infantum*, this practice should be tried. Two or three pints of water should be allowed to flow slowly into the rectum. If the hips are elevated it may be retained for some time, but is usually rapidly ejected. The water may be given either cold or warm; probably the latter would be better. During the last epidemic in Italy, Cantani used this method, which he calls *enteroclysis*, with great success. In each injection he gave tannic acid and, generally, laudanum.

Owing to the profuse serous discharges the blood becomes concentrated, and absorption takes place rapidly from the lymph-spaces. This it is which gives the shrunken puckered appearance to the features and skin of a patient in the collapse stage. To meet this, intravenous injections have been practised. My preceptor, Bovell, first practised the intravenous injections of milk in Toronto, in the epidemic of 1854. Less risky and equally efficacious is the subcutaneous injection of a saline solution. For this common salt should be used in the proportion of about four grammes to the litre. With rubber tubing, a canula from an aspirator, or even with a hypodermic needle, the warm solution may be allowed to run by pressure beneath the skin. It is rapidly absorbed, and the process may be continued until the pulse shows some sign of improvement. This is really a valuable method, thoroughly physiological, and should be tried in all severe cases.

In the stage of reaction special pains should be taken to regulate the diet and to guard against recurrences of the severe diarrhœa.

XIX. YELLOW FEVER.

Definition.—An acute febrile disease of tropical and subtropical countries, characterized by jaundice and hæmorrhages, and due to the action of a specific virus, the nature of which is yet unknown.

Etiology.—The disease prevails endemically in the West Indies and in certain sections of the Spanish Main. From these regions it occasionally extends and, under suitable conditions, prevails epidemically in the Southern States. Now and then it is brought to the large seaports of the Atlantic coast. Formerly it occurred extensively in the United States. In the latter part of the last century and the beginning of this, frightful epidemics prevailed in Philadelphia and other Northern cities. The epidemic of 1793, so graphically described by Matthew Carey, was the most serious that has ever prevailed in any city of the Middle States. The mortality, as given by Carey, during the months of August, September, October, and November, was 4,041, of whom 3,435 died in the months of September and October. The population of the city at the time was only 40,000. Epidemics occurred in the United States in 1797, 1798, 1799, and in 1802, when the disease prevailed slightly in Boston and extensively in Baltimore. In 1803 and 1805 it again appeared; then for many years the outbreaks were slight and localized. In 1853 the disease raged throughout the Southern States. In New Orleans alone there was a mortality of nearly eight thousand. In 1867 and 1873 there were moderately severe epidemics. In 1878 the last extensive epidemic occurred, chiefly in Louisiana, Alabama, and Mississippi. The total mortality was nearly sixteen thousand. In Europe it has occasionally gained a foothold, but there have been no wide-spread epidemics except in the Spanish ports. The disease exists on the west coast of Africa. It is sometimes carried to ports in Great Britain and France, but it has never extended into those countries. The history of the disease and its general symptomatology are exhaustively treated in the classical work of Réné La Roche.

Guitéras recognizes three areas of infection: (1) The focal zone in which the disease is never absent, including Havana, Vera Cruz, Rio, and other Spanish-American ports. (2) Perifocal zone or regions of periodic epidemics, including the ports of the tropical Atlantic in America and Africa. (3) The zone of accidental epidemics, between the parallels of 45° north and 35° south latitude.

The epidemics are invariably due to the introduction of the poison either by patients affected with the disease or through infected articles. Unquestionably the poison may be conveyed by fomites. Individuals of all ages and races are attacked. The negro is much less susceptible than the white, but he does not enjoy an immunity. Residents in southern countries, in which the disease is prevalent, are not so susceptible as strangers and temporary residents. Males are more frequently affected and the mortality is greater among them, owing probably to greater exposure.

Very young children usually escape; but in the epidemics of large cities the number under five attacked is large, since they constitute a considerable proportion of the population unprotected by previous attack. Guitéras states that the "foci of endemicity of yellow fever are essentially maintained by the creole infant population." Immunity is acquired by passing through an attack or by prolonged residence in a locality in which it is endemic. The statement so often made that the creoles are exempt from yellow fever has been abundantly disproved. They certainly are not so susceptible, but in severe epidemics they die in numbers. The evidence in favor of inherited immunity is not conclusive.

Conditions favoring the Development of Epidemics.—Yellow fever is a disease of the sea-coast, and rarely prevails in regions with an elevation above one thousand feet. Its ravages are most serious in cities, particularly when the sanitary conditions are unfavorable. It is always most severe in the badly drained, unhealthy portions of a city, where the population is crowded together in ill-ventilated, badly drained houses. The disease prevails during the hot season. In Havana the death-rate is greatest during the months of June, July, and August. The epidemics in the United States have always been in the summer and autumn months.

The specific germ of the disease has not yet been discovered. Sternberg, in his last report to the United States Government, concludes that the specific cause of yellow fever has not yet been demonstrated. With this statement Cornil and Babes * agree, and they do not accept the organisms described by Fréire, Carmona, and Gibier.

Morbid Anatomy.—The skin is more or less jaundiced. Cutaneous hæmorrhages may be present. No specific or distinctive internal lesions have been found. The blood-serum contains hæmoglobin, owing to destruction of the red cells, just as in pernicious malaria. The heart sometimes, not invariably, shows fatty change; the stomach presents more or less hyperæmia of the mucosa with catarrhal swelling. It contains the material which, ejected during life, is known as the *black vomit*. The essential ingredient in this is transformed blood-pigment. In the two specimens which I have had an opportunity of examining it differed in no respect from the material found in other affections associated with hæmatemesis. There is no proof that this black material depends upon the growth of a micro-organism. The liver is usually of a pale yellow or brownish-yellow color, and the cells are in various stages of fatty degeneration. From the date of Louis's observations at Gibraltar in 1828, the appearances of this organ have been very carefully studied, and some have thought the changes in it to be characteristic. Councilman has described remarkable appearances in the liver-cells which he believes are distinctive and peculiar. Fatty degeneration and regions of necrosis are present in all cases. The kidneys often show traces of diffuse nephritis. The epi-

* Les Bactéries, 1890.

thelium of the convoluted tubules is swollen and very granular; there may also be necrotic changes. In both liver and kidneys bacteria of various sorts have been described.

Symptoms.—The incubation is usually three or four days, but it may be less than twenty-four hours and prolonged to seven days. The onset is sudden; as a rule, without preliminary symptoms. An initial chill is common, and with it are usually associated headache and pains in the back and limbs. The fever rises rapidly and the skin feels very hot and dry. The face is flushed; the tongue furred, but moist; the throat sore. Nausea and vomiting are present, and become more intense on the second or third day. The bowels are usually constipated. The urine is reduced in amount and may be albuminous from the outset. The pulse, at first, has the usual febrile characters, but quickly becomes feeble and, as the jaundice develops, may become slow. This stage of invasion, or the *febrile stage*, lasts from a few hours to two or three days. It is succeeded by a remission, or, as it has sometimes been called, the *stage of calm*, during which the temperature falls and the severity of the symptoms abates. In favorable cases the fever now subsides and convalescence sets in. In such cases jaundice may not develop. In the third stage, or that of the *febrile reaction*, the temperature rises again and the symptoms become aggravated. The jaundice develops rapidly, the vomiting increases, and, in a considerable proportion of the cases, bl vomit occurs. This consists of blood and gastric mucus altered acid juices of the stomach. Though usually regarded as distinctive and characteristic of the disease, material identical with it is brought up under other febrile conditions in which vomiting of blood occurs. Altered blood-corpuscles, epithelial cells, portions of food, and various fungi are found in the fluid. The vomiting may be accompanied by great abdominal pain. The stools are often tarry from the presence of altered blood. In mild cases the vomiting ceases during the first stage of the disease. Black vomit is not necessarily a fatal symptom, though it is present only in the severer cases of the disease. Jaundice occurs in a limited number of the cases which recover, and is present in almost all the fatal cases. From the character of the disease it is probably hæmatogenous in its origin. Bleeding may occur from the kidneys or from the gums, and hæmorrhages into the skin are not uncommon. As would be expected in a fever of this nature, the urine is albuminous; the amount varying a good deal with the intensity of the fever, and with the grade of jaundice. Febrile icterus, from whatever cause, is almost invariably associated with albuminuria and tube-casts, and the evidences of a diffuse nephritis.

Relapses occasionally occur. Among the varieties of the disease it is important to recognize the mild cases. These are characterized by slight fever, continuing for one or two days, and succeeded by a rapid convalescence. Such cases would not be recognized as yellow fever in the absence of a prevailing epidemic. Cases of greater severity have high fever and

the features of the disease are well marked—vomiting, prostration, and hæmorrhages. And lastly there are malignant cases in which the patient is overwhelmed by the intensity of the fever, and death takes place in two or three days.

In severe cases convalescence may be complicated by the occurrence of parotitis, abscesses in various parts of the body, and diarrhœa. An attack confers an immunity which persists, as a rule, through life.

Diagnosis.—Mild cases, and even severe cases in the early period of an epidemic, are very difficult to recognize. The disease simulates closely, and may be mistaken for ordinary malarial remittent fever. It is not uncommon for physicians, in regions in which yellow fever is occasionally epidemic, to call the milder cases malarial fever, reserving the name of yellow fever for the severer forms with jaundice and black vomit. The only disease with which these cases could be confounded is malaria in its remittent and pernicious forms. But yellow fever can now be definitely and at once separated by the examination of the blood. Twice in Philadelphia I was sent for to determine whether a patient, freshly arrived in the city from the South, had yellow fever or pernicious malaria; and I was able in both instances, by finding Lavaran's organisms in the blood, to pronounce definitely upon the nature of the disease. The clinical picture in certain cases of malarial remittent and yellow fever may be almost identical. The presence of albumen in the urine, upon which some writers lay such stress as a distinguishing feature in yellow fever, is far too common a symptom in all forms of malaria to be worth much as a guide. Guitéras states that there may be difficulty for a time in recognizing the difference between mild cases of thermic fever and yellow fever.

Prognosis.—In its graver forms, yellow fever is one of the most fatal of epidemic diseases. The mortality has ranged, in various epidemics, from 15 to 85 per cent. In heavy drinkers and those who have been exposed to hardships the death-rate is much higher than among the better classes. In the epidemic of 1878, in New Orleans, while the mortality in hospitals was over 50 per cent of the white and 21 per cent of the colored patients, in private practice the mortality was not more than 10 per cent among the white patients. Favorable symptoms are a low grade of fever, slight jaundice, absence of hæmorrhages, and a free secretion of urine. If the temperature rises above 103° or 104° during the first two days, the outlook is serious. Black vomit is not an invariably fatal symptom. Cases with suppression of urine, delirium, coma, and convulsions rarely recover.

Prophylaxis.—The measures to be taken are—

(*a*) "Exclusion of the exotic germ of the disease by the sanitary supervision, at the port of departure, of ships sailing from infected ports, and thorough disinfection at the port of arrival, when there is evidence or reasonable suspicion that they are infected; (*b*) isolation of the sick on ship-

board, at quarantine stations, and, so far as practicable, in recently infected places; (*c*) disinfection of excreta, and of the clothing and bedding used by the sick, and of localities into which cases have been introduced, or which have become infected in any way; (*d*) depopulation of infected places—i. e., the removal of all susceptible persons whose presence is not necessary for the care of the sick" (Sternberg). During an epidemic, individuals who must remain in the locality should avoid the regions in which the disease prevails most; they should live temperately, avoiding all excesses, and should be careful not to get overheated, either in the sun or by exercise. It is very doubtful whether the preventive inoculations introduced by Fréire in Brazil and Carmona in Mexico are of any value.

Treatment.—Careful nursing and a symptomatic plan of treatment probably give the best results. Bleeding has long since been abandoned. How much patients will stand in this disease is illustrated by Rush's practice, which was of the most heroic character. He says: "From a newly arrived Englishman I took 144 ounces, at twelve bleedings, in six days; four were in twenty-four hours. I gave within the course of the same six days nearly 150 grains of calomel, with the usual proportions of jalap and gamboge"* With the courage of his convictions this modern Sangrado himself submitted to two bleedings in one day, and had his infant of six weeks old bled twice. Neither emetics nor purgatives are now employed. Of special remedies quinine is warmly recommended, and, when hæmorrhage sets in, the perchloride of iron. Digitalis, aconite, and jaborandi have been employed. Sternberg advises the following mixture: Bicarbonate of soda, 150 grains; bichloride of mercury, $\frac{1}{8}$ grain; pure water, 1 quart. Three tablespoonfuls to be given every hour. This is given on the view that the specific agent is in the intestine, and that its growth may possibly be restrained by this antacid and antiseptic mixture. The fever is best treated by hydrotherapy. There are several reports of the good effects of cold baths, sponging, and the application of ice-cold water to the head and the extremities in this disease. Vomiting is a very difficult symptom to control. Morphia hypodermically and ice in small quantities are probably the best remedies. Medicines given by the mouth for this purpose are said to be rarely efficacious.

We have no reliable medicine which can be depended upon to check the hæmorrhages. Ergot and acetate of lead and opium are recommended. The uræmic symptoms are best treated by the hot bath. Stimulants should be given freely during the second stage, when the heart's action becomes feeble and there is a tendency to collapse. The patient should be carefully fed; but when the vomiting is incessant it is best not to irritate the stomach, but to give nutritive enemata until the gastric irritation is allayed.

* Manuscript letter to Redman Coxe.

XX. DYSENTERY.

Definition.—Under this clinical term several different forms of intestinal flux are described, which are characterized by frequent stools, and in the acute stage are accompanied by tormina and tenesmus. Anatomically there are inflammation and usually ulceration of the large bowel.

Etiology.—Dysentery is one of the four great epidemic diseases of the world. In the tropics it destroys more lives than cholera, and it has been more fatal to armies than powder and shot.

While especially severe in the tropics, sporadic cases constantly occur in more temperate climates, and under favoring circumstances epidemics are found even in the more northern countries, such as Canada and Norway. It has become less frequent of late years, owing to improved sanitary conditions. The statistics of the Montreal General Hospital, for the twenty years ending May 1, 1889, show a remarkable decrease in the disease. In the decade ending May, 1879, 150 cases were admitted; whereas in the last ten years there have been only 31 cases admitted. There has been a similar decrease at the Pennsylvania Hospital.

In the Southern cities of this country dysentery is more prevalent; even when not epidemic, sporadic cases are common. In Baltimore it prevails every summer, and has on several occasions been epidemic.

Epidemics of dysentery have occurred in the United States for more than a century, and Woodward has collected the data which show the various outbreaks. Perhaps the most serious was that which prevailed in various localities from 1847 to 1856. During the war of secession the disease existed to an alarming extent in both armies. According to Woodward's report,* there were in the Federal service in all 259,071 cases of acute and 28,451 cases of chronic dysentery. Probably a considerable proportion of the 182,586 cases of chronic diarrhœa should also come in this category. The decennial census reports since 1850 show a progressive decrease in the total number of deaths from this disease. It prevails most extensively in the summer and autumn. Sudden changes of temperature appear more harmful than variations in moisture. The effluvia from decomposing animal matter have been thought by some to predispose to or even to cause the disease. That dysenteric affections are more frequent in malarial localities has long been known, and is probably connected with external conditions favoring their development. With reference to the influence of drinking-water, Woodward is doubtless correct in stating that the effects of dissolved mineral matters have been greatly exaggerated. On the other hand, from the days of the old Greek physicians, it has been held that the impurities in the stagnant water of marshy districts and

* Medical and Surgical History of the War of the Rebellion, Medical, vol. ii; the most exhaustive treatise extant on intestinal fluxes—an enduring monument to the industry and ability of the author.

ponds may give rise to diarrhœa and dysentery. Here, however, it is probably not the vegetable impurities which are directly causative, but the organic matter renders the water a more favorable medium for the development of organisms which may cause disease.

Dyspeptic conditions, particularly those caused by the ingestion of bad food and unripe fruit, seem to predispose to the disease. Great stress has been laid by German authorities on the importance of constipation as a causal factor in dysentery.

Dysentery occurs at all ages. There is no race immunity. The contagiousness of the disease is doubtful. The experience of the civil war is decidedly against it, but the possibility, as with typhoid fever, must be acknowledged.

Clinical Forms.—(*a*) **Acute Catarrhal Dysentery.**—This may occur sporadically or endemically, and is the variety most frequently found in temperate climates.

Morbid Anatomy.—The lesions are confined to the large bowel, and sometimes the ileum also is involved. The mucous membrane is injected, swollen, and often covered with tenacious blood-stained mucus. The most striking feature is the enlargement of the solitary follicles, which stand out prominently from the mucous membrane. In very acute forms, as in children, the picture is that of an acute follicular colitis. In more protracted cases the follicles suppurate or are capped with an area of necrotic tissue. In other instances the sloughs have separated and the entire colon presents numerous ulcers, most of which have developed from the follicles, and others have resulted from necrosis and sloughing of the intervening tissue.

Symptoms.—There may be preliminary dyspepsia or slight pains in the abdomen. Chills are rare. Diarrhœa is the most constant initial symptom, and at first is not painful. Usually within thirty-six hours the characteristic features of the disease develop—abdominal pain of a colicky, griping character, frequent stools, which are passed with straining and tenesmus; the constitutional disturbance is variable, and in mild cases may be slight. The temperature range is not high, but at the outset the fever may rise to 102° or 103°. The tongue is furred and moist, and as the disease progresses becomes red and glazed. Nausea and vomiting may be present, but as a rule the patient retains nourishment. The constant desire to go to stool and the straining or tenesmus are the most distressing symptoms. The abdomen may be flat and hard. The thirst is often excessive. The stools in this variety of dysentery have the following characters: During the first twenty-four or forty-eight hours they consist of more or less clear mucus and blood mixed with small fæcal scybala. After this they become purely gelatinous and bloody, and are small and frequent, from fifteen to two hundred in twenty-four hours, according to the severity of the case. About the end of the first week the mucus becomes opaque, the proportion of blood diminishes, and grayish or brownish shreddy material

appears in the stools, which become gradually reduced in frequency. Some of the stools at this time may be wholly composed of a greenish pultaceous material and mucus. As the disease subsides, fæcal matter again appears in the stools, increasing in amount until fully formed fæces are passed, containing no mucus or blood. Microscopical examination of the glairy bloody stools shows red blood-corpuscles, few or many leucocytes, and constantly large, swollen, round or oval epithelioid cells, containing fat-drops and vacuoles. Bacteria are scarce; occasionally the *cercomonas intestinalis* is seen in large numbers.

Course of the Disease.—The milder cases run a course, as Flint has shown, of about eight days; severer ones rarely terminate within four weeks. Recovery may be imperfect, and the affection occasionally becomes chronic. In this form the complications are not numerous; peritonitis and liver abscess are extremely rare. Except in young children, a majority of the cases terminate favorably.

(*b*) **Tropical Dysentery—Amœbic Dysentery.**—This form of intestinal flux is characterized by irregular diarrhœa and the constant presence in the stools of the *amœba coli* (Lösch), *amœba dysenteriæ* (Councilman and Lafleur). It is this variety which prevails extensively in the tropical and subtropical regions, and which proves so fatal in epidemic form. The amœba is a unicellular, protoplasmic, motile organism, from ten to twenty micromillimetres in diameter, consisting of a clear outer zone, ectosarc, and a granular inner zone, endosarc, containing a nucleus and one or more vacuoles. It was first described by Lambl in 1859, and subsequently by Lösch, who considered it the cause of the disease. In the endemic dysentery of Egypt, Kartulis, in 1883, found these amœbæ constantly in the stools, in the intestines, and in the liver abscesses. He was afterward enabled to cultivate them in straw infusion, and to produce the disease artificially in cats and dogs. In 1890 I reported a case of dysentery with abscess of the liver originating in Panama, in which the amœbæ were found in the stools and in the pus from the abscess; and lately Councilman and Lafleur* have described the clinical features and anatomical lesions in a series of cases of this form of dysentery in my wards. Dock, in Galveston, has demonstrated their presence in a number of cases, and Musser has found them in Philadelphia. The disease is very common in tropical and subtropical countries. It is, however, found more or less widely distributed throughout Europe and North America. The sources of infection are not known, but it seems probable that one of them is drinking-water.

Morbid Anatomy.—The lesions are found in the large intestine, sometimes in the lower portion of the ileum. Abscess of the liver is a common sequence. Perforation into the right lung is not infrequent.

Intestines.—The lesions consist of ulceration, produced by preceding

* Johns Hopkins Hospital Reports, vol. ii.

infiltration, general or local, of the submucosa, the general infiltration being due to an œdematous condition, the local to multiplication of the fixed cells of the tissue. In the earliest stage these local infiltrations appear as hemispherical elevations above the general level of the mucosa. The mucous membrane over these soon becomes necrotic and is cast off, exposing the infiltrated submucous tissue as a grayish-yellow gelatinous mass, which at first forms the floor of the ulcer, but is subsequently cast off as a slough.

The individual ulcers are round, oval, or irregular, with infiltrated, undermined edges. The visible aperture is often small compared to the loss of tissue beneath it, the ulcers undermining the mucosa, coalescing, and forming sinuous tracts bridged over by apparently normal mucous membrane. According to the stage at which the lesions are observed, the floor of the ulcer may be formed by the submucous, the muscular, or the serous coat of the intestine. The ulceration may affect the whole or some portion only of the large intestine, particularly the cæcum, the hepatic and sigmoid flexures, and the rectum. In severe cases the whole of the intestine is much thickened and riddled with ulcers, with only here and there islands of intact mucous membrane.

The disease advances by progressive infiltration of the connective-tissue layers of the intestine, which produces necrosis of the overlying structures. Thus, in severe cases there may be in different parts of the bowel sloughing *en masse* of the mucosa or of the muscularis, and the same process is observed, but not so conspicuously, in the less severe forms.

In some cases a secondary diphtheritic inflammation complicates the original lesions.

Healing takes place by the gradual formation of fibrous tissue in the floor and at the edges of the ulcers, which may ultimately result in partial and irregular strictures of the bowel.

Microscopical examination shows a notable absence of the products of purulent inflammation. In the infiltrated tissues polynuclear leucocytes are seldom found, and never constitute purulent collections. On the other hand, there is proliferation of the fixed connective-tissue cells. Amœbæ are found more or less abundantly in the tissues at the base of and around the ulcers, in the lymphatic spaces, and occasionally in the blood-vessels.

The lesions in the *liver* are of two kinds: firstly, local necroses of the parenchyma, scattered throughout the liver and possibly due to the action of chemical products of the amœbæ; and, secondly, abscesses. These may be single or multiple. When single they are generally in the right lobe, either toward the convex surface near its diaphragmatic attachment, or on the concave surface in proximity to the bowel. Multiple abscesses are small and generally superficial. In an early stage the abscesses are grayish-yellow, with sharply defined contours, and contain a spongy necrotic material, with more or less fluid in its interstices. The larger abscesses have

ragged necrotic walls, and contain a more or less viscid, greenish-yellow or reddish-yellow purulent material mixed with blood and shreds of liver-tissue. The older abscesses have fibrous walls of a dense, almost cartilaginous toughness. A section of the abscess wall shows an inner necrotic zone, a middle zone in which there is great proliferation of the connective-tissue cells and compression and atrophy of the liver-cells, and an outer zone of intense hyperæmia. There is the same absence of purulent inflammation as in the intestine, except in those cases in which a secondary infection with pyogenic organisms has taken place. The material from the abscess cavity shows chiefly fatty and granular detritus, few cellular elements, and more or less numerous amœbæ. Amœbæ are also found in the abscess walls, chiefly in the inner necrotic zone. Cultures are usually sterile. Lesions in the lungs are seen when an abscess of the liver—as so frequently happens—points toward the diaphragm and extends by continuity through it into the lower lobe of the right lung. The gross and microscopical appearances are similar to those of the liver.

Symptoms.—The onset may be sudden, as in catarrhal dysentery, or gradual, beginning as a trifling and perhaps transient diarrhœa. In severe gangrenous cases the abrupt onset is more common. The subsequent course is a very irregular diarrhœa, marked by exacerbations and intermissions, and progressive loss of strength and flesh. There is moderate fever as a rule, but many cases are afebrile throughout the greater part of their course. Abdominal pain and tenesmus are frequently present at the onset, especially in severe cases, but may be entirely absent, and vomiting and nausea are only occasionally observed. The stools vary very much in frequency and appearance in different cases and at different periods in the same cases. They may be very frequent, bloody, and mucoid at the outset, as in catarrhal dysentery; but their main characteristic, when the disease is well established, is fluidity. From six to twelve yellowish-gray liquid stools, containing mucus and occasionally blood in varying proportions, are passed daily for weeks. Actively moving amœbæ are found in these stools, more abundantly during exacerbations of the diarrhœa, and disappear gradually as the stools become formed.

Abscess of the liver, and especially of the liver and lung, is a frequent and formidable complication. In India it occurs once in every four or five cases.

The duration of the disease in uncomplicated cases varies from six to twelve weeks. Recovery is tedious, owing to anæmia and muscular weakness, often delayed by relapses, and there is in all cases a constant tendency to chronicity. The mortality is much higher than in catarrhal dysentery. A fatal issue is due either to the initial gravity of the intestinal lesions, to exhaustion in prolonged cases, or to involvement of the liver.

(*c*) **Diphtheritic Dysentery.**—A form of colitis or entero-colitis in which areas of necrosis occur in the mucous membranes, which on sepa-

ration leave ulcers. This occurs: (*a*) As a *primary* disease coming on acutely and sometimes proving fatal. In its milder grades the tops of the folds of the colon are capped with a thin, yellow exudate. In severer forms the colon is enormously enlarged, the walls are thickened, stiff, and infiltrated, and the mucosa, from the ileo-cæcal valve to the rectum, represented by a tough, yellowish material, in which on section no trace of the glandular elements can be seen. It is an extensive necrosis of the mucosa. There are cases in which this necrosis is superficial, involving only the upper layers of the mucous membrane; but in the most advanced forms it may be, as in the description by Rokitansky, "a black, rotten, friable, charred mass." The areas of necrosis may be more localized, and large sloughs are formed which may be a half to three fourths of an inch in thickness and extend to the serosa. There are instances in which this condition is confined to the lower portion of the large bowel. A sailor from the Mediterranean was admitted to the Montreal General Hospital under my care with symptoms resembling typhoid fever. The autopsy showed enormous sloughs in the rectum and in the sigmoid flexure, but scarcely any disease in the transverse or ascending colon. In cases which last for many weeks the sloughs separate and may be thrown off, sometimes in large tubular pieces.

(*b*) *Secondary Diphtheritic Dysentery.*—This occurs as a terminal event in many acute and chronic diseases. It is not infrequent in chronic heart affections, in Bright's disease, and in cachectic states generally. In acute diseases it is, as pointed out by Bristowe, most frequently associated with pneumonia. Anatomically there may be only a thin, superficial infiltration of the upper layer of the mucosa in localized regions, particularly along the ridges and folds of the colon, often extending into the ileum. In severer forms the entire mucosa may be involved and necrotic, sometimes having a rough, granular appearance. In the secondary colitis of pneumonia the exudation may be pseudo-membranous and form a firm, thin, white pellicle which seems to lie upon, not within, the mucous membrane.

Symptoms.—The clinical features of diphtheritic dysentery are very varied. In the acute *primary* cases the patient from the outset is often extremely ill, with high fever, great prostration, pain in the abdomen, and frequent discharges. Delirium may be early and the clinical features may closely resemble severe typhoid. I have, on more than one occasion, known this mistake to be made. The abdomen is distended and often tender. The discharges are frequent and diarrhœal in character, and tenesmus may not be a striking symptom. Blood and mucus may be found early, but are not such constant features as in the follicular disease. This primary form is very fatal, but the sloughs may separate and the condition become chronic. In the *secondary* form there may have been no symptoms to attract attention to the large bowel. In a majority of the cases the patient has a diarrhœa—three, four, or more movements in the

day, which are often profuse and weakening. A little blood and mucus may be passed at first, but they are not specially characteristic elements in the stools.

In all forms of dysentery death usually results from asthenia. The pulse becomes weaker and more rapid, the tongue dry, the face pinched, the skin cool and covered with sweat, and the patient falls into a drowsy, torpid condition. Consciousness may be retained until the last, but in the protracted cases there is a low delirium deepening into collapse.

(*d*) **Chronic Dysentery.**—This usually succeeds an acute attack, though the amœbic form may be subacute from the outset and not present an acute period. Anatomical changes in the large intestine in chronic dysentery are variable. There may be no ulceration, and the entire mucosa presents a rough, irregular puckered appearance, in places slate-gray or blackish in color. The submucosa is thickened and the muscular coats are hypertrophied. There may be cystic degeneration of the glandular elements, as is beautifully figured in Woodward's volume.

Ulcers are usually present, often extensive and deeply pigmented, in places perhaps healing. The submucous and muscular coats are thickened and the calibre of the bowel may be reduced. Stricture, however, is very rare.

The *symptoms* of chronic dysentery are by no means definite, and it is not always possible to separate the cases from those of chronic diarrhœa. Many of the characteristic symptoms of the acute disease are absent. Tenesmus and severe griping pains rarely occur except in acute exacerbations. The character of the stools varies very much. Blood and necrotic shreddy tissue are not often found. Mucus is passed in variable amounts. On a mixed diet the fæces are thin, often frothy, and contain particles of food. The motions vary from four or five to twelve or more in the twenty-four hours. There are cases in which marked constipation alternates with attacks of diarrhœa, and scybala may be passed with much mucus. In many cases the fæces have a semi-fluid consistency, and a yellowish or brown color depending on the amount of bile. Fragments of undigested food may be found, and the discharges have the character of what is termed a lienteric diarrhœa. Indeed, variations in the bile and in the food give at once corresponding variations in the character of the stools. In chronic dysentery recurrences are common in which blood and mucus again appear in the stools, accompanied perhaps by pus. Flatulence is in some cases distressing, and there is always more or less tenderness along the course of the colon. The appetite is capricious, the digestion disordered, and unless the patient is on a strictly regulated diet the number of stools is greatly increased. The tongue is not often furred; it is more commonly red, glazed, and beefy, and becomes dry and cracked toward the end in protracted cases. There is always anæmia and the emaciation may be extreme; with the exception of gastric cancer, we rarely see such ghastly faces as in patients with prolonged dysentery.

The complications are those already referred to in the acute form. The greater debility renders the patient more liable to the intercurrent affections, such as pneumonia and tuberculosis. Ulceration of the cornea was frequently noted during the civil war.

Complications and Sequelæ.—A local peritonitis may arise by extension, or a diffuse inflammation may follow perforation, which is usually fatal. When this occurs about the cæcal region, perityphlitis results; when low down in the rectum, periproctitis. In one hundred and eight autopsies collected by Woodward perforation occurred in eleven. By far the most serious complication is abscess of the liver, which occurs frequently in the tropics and is not very uncommon in this country. It was not, however, a frequent complication in dysentery during the civil war. In this latitude it is certainly not uncommon, as we have had five cases, within two years, in the Johns Hopkins Hospital. It usually comes on insidiously. The symptoms will be discussed in connection with hepatic abscess.

It is stated that malaria is a complication, but with one exception the cases which I have seen with intermittent pyrexia were invariably associated with suppuration. In extensive epidemics, however, Woodward states that cases of ordinary dysentery occur associated with all the phenomena of malaria. With reference to typhoid fever, as a complication, this author mentions that the combination was exceedingly frequent during the civil war, and characteristic lesions of both diseases coexisted. In civil practice it must be extremely rare

Sydenham noted that dysentery was sometimes associated with rheumatic pains, and in certain epidemics joint swellings have been especially prevalent. They are probably not of the nature of true rheumatism, but are rather analogous to gonorrhœal arthritis. In severe, protracted cases there may be pleurisy, pericarditis, endocarditis, and occasionally pyæmic manifestations, among which may be mentioned pylephlebitis. Chronic Bright's disease is also an occasional sequel. In protracted cases there may be an anæmic œdema. An interesting sequel of dysentery is paralysis. Woodward reports eight cases. Weir Mitchell mentions it as not uncommon, occurring chiefly in the form of paraplegia. As in other acute fevers, this is due to a neuritis.* Intestinal stricture is a rare sequence—so rare that no case was reported at the Surgeon-General's office during the war. Among the sequelæ of chronic dysentery, in persons who have recovered a certain measure of health, may be mentioned persistent dyspepsia and irritability of the bowels.

Diagnosis.—The recognition of the acute follicular form is easy; the frequency of the passages, the presence of blood and mucus, and the tenesmus forming a very characteristic picture. Local affections of the rectum, particularly syphilis and epithelioma, may produce tenesmus with

* Pugibet, Revue de Médecine, February, 1888.

the passage of mucoid and bloody stools. The acute diphtheritic form, coming on with great intensity and with severe constitutional disturbances, is not infrequently mistaken for typhoid fever, to which indeed in many cases the resemblance is extremely close. The higher grade of fever, the more pronounced intestinal symptoms, the presence, particularly in the early stage, of a small amount of blood in the stools, the absence of enlargement of the spleen and the rose rash should lead to a correct diagnosis. In the amœbic form the diagnosis can readily be made by examination of the stools. A characteristic feature of these cases is their irregular, chronic course. A patient may be about and in fairly good condition, with well-formed stools and very slight intestinal disturbance, in whose fæces the amœbæ may still be discovered, and in whom the disease is at any time likely to recur with intensity. In some cases, complicated by abscess of the liver and lung discharging through a bronchus, the diagnosis may rest on the detection of amœbæ in the sputa, when they cannot be found in the stools owing to the latency of the intestinal disturbance. Three such cases occurred in my wards in 1890.*

Treatment.—Flint has shown that sporadic dysentery is, in its slighter grades at least, a self-limited disease, which runs its course in eight or nine days. Reading a report of his cases, one is struck, however, with their comparative mildness.

The enormous surface involved, amounting to many square feet, the constant presence of irritating particles of food, and the impossibility of getting absolute rest, are conditions which render the treatment of dysentery peculiarly difficult. Moreover, in the severer cases, when necrosis of the mucosa has occurred, ulceration necessarily follows, and cannot in any way be obviated. When a case is seen early, particularly if there has been constipation, a saline purge should be given. The free watery evacuations produced by a dose of salts cleanse the large bowel with the least possible irritation, and if necessary, in the course of the disease, particularly if scybala are present, the dose may be repeated. Purgatives are, as a rule, objectionable, and the profession has largely given up their use. Of medicines given by the mouth which are supposed to have a direct effect upon the disease, ipecacuanha still maintains its reputation in the tropics. It did not, however, prove satisfactory during the civil war; nor can I say that in cases of sporadic dysentery I have ever seen the marked effect described by the Anglo-Indian surgeons. The usual method of administration is to give a preliminary dose of opium, in the form of laudanum or morphia, and half an hour after from twenty to sixty grains of ipecacuanha. If rejected by vomiting, the dose is repeated in a few hours.

Minute doses of corrosive sublimate, one hundredth of a grain every two hours, are warmly recommended by Ringer. Large doses of bismuth, half a drachm to a drachm every two hours, so that the patient may take

* For details see monograph of Councilman and Lafleur.

from twelve to fifteen drachms in a day, have in many cases had a beneficial effect. To do good it must be given in large doses, as recommended by Monneret, who gave as high as seventy grammes a day. It certainly is more useful in the chronic than the acute cases. It is best given alone. Opium is an invaluable remedy for the relief of the pain and to quiet the peristalsis. It should be given as morphia, hypodermically, according to the needs of the case.

The treatment of dysentery by topical applications is by far the most rational plan. A serious obstacle, however, in the acute cases, is the extreme irritability of the rectum and the tenesmus which follows any attempt to irrigate the colon. A preliminary cocaine suppository or the injection of a small quantity of the four-per-cent solution will sometimes relieve this, and then with a long tube the solution can be allowed to flow in slowly. The patient should be in the dorsal position with a pillow under the hips, so as to get the effect of gravitation. Water at the temperature of 100° is very soothing, but the irritability of the bowel is such that large quantities can rarely be retained for any time. When the acute symptoms subside, the injections are better borne. Various astringents may be used—alum, acetate of lead, sulphate of zinc and copper, and nitrate of silver. Of these remedies the nitrate of silver is the best, though I think not in very acute cases. In the chronic form it is perhaps the most satisfactory method of treatment which we have. It is useless to give it in the small injections of two or three ounces with one to two grains of the salt to the ounce. It must be a large irrigating injection, which will reach all parts of the colon. This plan was introduced by Hare, of Edinburgh, and is highly recommended by Stephen MacKenzie and H. C. Wood. The solution must be fairly strong, twenty to thirty grains to the pint, and if possible from three to six pints of fluid must be injected. To begin with it is well to use not more than a drachm to the two pints or two and a half pints, and to let the warm fluid run in slowly through a tube passed far into the bowel. It is at times intensely painful and is rejected at once. In the cases of amœbic dysentery we have been using at the Johns Hopkins Hospital with great benefit warm injections of quinine in strength of 1 to 5,000, 1 to 2,500, and 1 to 1,000. The amœbæ are rapidly destroyed by it. These large injections are not without a certain degree of danger. Brayton Ball reports the case of a child in whom general peritonitis followed the injections. I have never seen any ill effects, even with the very large amounts. When there is not much tenesmus, a small injection of thin starch with half a drachm to a drachm of laudanum gives great relief, but for the tormina and tenesmus, the two most distressing symptoms, a hypodermic of morphia is the only satisfactory remedy. Local applications to the abdomen, in the form of light poultices or turpentine stupes, are very grateful.

The diet in acute cases must be restricted to milk, whey, and broths,

and during convalescence the greatest care must be taken to provide only the most digestible articles of food. In chronic dysentery, diet is perhaps the most important element in the treatment. The number of stools can frequently be reduced from ten or twelve in the day to two or three, by placing the patient in bed and restricting the diet. Many cases do well on milk alone, but the stools should be carefully watched and the amount limited to that which can be digested. If curds appear, or if much oily matter is seen on microscopical examination, it is best to reduce the amount of milk and to supplement it with beef-juice or, better still, egg-albumen. The large doses of bismuth seem specially suitable in the chronic cases, and the injections of nitrate of silver, in the way already mentioned, should always be given a trial.

XXI. MALARIAL FEVER.

Definition.—An infectious disease characterized by: (*a*) paroxysms of intermittent fever of quotidian, tertian, or quartan type; (*b*) a continued fever with marked remissions; (*c*) certain pernicious, rapidly fatal forms; and (*d*) a chronic cachexia, with anæmia and an enlarged spleen.

With the disease are invariably associated the hæmatozoa described by Laveran.

Etiology.—(1) **Geographical Distribution.**—In Europe, southern Russia and certain parts of Italy are now the chief seats of the disease. It is not widely prevalent in Germany, France, or England, and the foci of epidemics are becoming yearly more restricted. In America it is now rare on the Atlantic coast above the latitude of Philadelphia. From New England, where it once prevailed extensively, it has gradually disappeared, but there has of late years been a slight return in some places. In the city of New York genuine malaria is rare except as an imported disease. In Philadelphia and along the valleys of the Delaware and Schuylkill Rivers, formerly hot-beds of malaria, the disease has become much restricted. Except in the low-lying southern portions of the city it rarely develops, and the majority of cases admitted into hospital are of the poorer class, who have returned from picking cranberries and peaches in Delaware and New Jersey. In Baltimore a few cases develop in the autumn, but a majority of the patients seeking relief are from the outlying districts and one or two of the inlets of Chesapeake Bay. Though prevalent in certain regions on this bay, the disease is yearly becoming less widespread and less severe. In the Southern States there are on the seaboard many isolated regions in which malaria prevails; but here, too, there has everywhere been a marked diminution in the prevalence and intensity of the disease. W. W. Johnston states that in the Gulf district there are places in which the disease is increasing. The percentage of cases admitted to the Marine Hospital Service in 1876 was 18·4, and 23·4 in 1887.

But this may be due to the development of the shipping trade and to the greater number of sailors who carry the infection from the West Indian ports, and those of Mexico and Central America.

In the interior of Louisiana, Mississippi, Arkansas, and Texas malaria is endemic, and the severe types are not infrequent. At irregular periods epidemics of the most severe forms occur.

In the Western and Northwestern States malaria is almost unknown. It is rare on the Pacific coast. In the region of the Great Lakes malaria prevails only in the Lake Erie and Lake St. Clair regions. It has practically disappeared from Lake Ontario, whereas in the upper Huron and Lake Superior basins it is unknown. The St. Lawrence River region remains free from the disease. In Montreal a patient with malaria is invariably questioned as to his latest residence.

(2) **Telluric Conditions.**—The importance of the state of the soil in the etiology of malaria is universally recognized. It is seen particularly in low, marshy regions which have an abundant vegetable growth. Estuaries, badly drained, low-lying districts, the course of old river-beds, tracts of land which are rich in vegetable matter, and particularly districts such as the Roman Campagna, which have been allowed to fall out of cultivation, are favorite localities for the development of the malarial poison. These conditions are most frequently found, of course, in tropical and subtropical regions, but nothing can be truer than the fact that reeking marshes of the most pestilent appearance may be entirely devoid of the poison, and the disappearance of the disease from a locality is not necessarily associated with any material improvement in the condition of the marshes or of the soil. Thus, in New England and in parts of western Canada, in which malaria formerly was very prevalent, the increased salubrity is usually attributed to the clearing of the forests and the better drainage of the ground; but these improvements alone can scarcely explain the disappearance, since in many districts there are marshy tracts and low-lying lands in every respect like those in which, even at the same latitude, the disease still prevails. Compare, for example, a swampy tract on the northern shore of Lake Erie and a similar tract on the southern shore of Lake Ontario; the flora and fauna of the two districts are practically identical, but in the former the conditions under which the malarial virus develops still exist, whereas in the latter they have gradually disappeared. In short, it is impossible to ascertain from the nature of the soil and climate in any given place whether it is malarial or not. In the absence of accurate knowledge as to the habitat of the hæmatozoa, the only means of deciding this point is by noticing the effect of residence in such a place on the human subject, preferably one of the Caucasian race.

(3) **Season.**—Even in the tropics, where malaria constantly prevails, there are minimal and maximal periods; the former corresponding to the summer and winter, the latter to the spring and autumn months. In temperate regions, like the central Atlantic States, there are only a few

cases in the spring, usually in the month of May, and a large number of cases in September and October, and sometimes in November. In the tropics, too, the cases are most numerous in the autumn months.

(4) **Meteorological Conditions.**—(*a*) *Heat.*—A tolerably high temperature is one of the essential conditions for the development of the virus. It is more prevalent after prolonged hot summers.

(*b*) *Moisture.*—In the tropics the malarial fevers are most prevalent in the rainy seasons. In the temperate climates the relation between the rainfall and malaria is not so clear, and cases are more numerous after a dry summer; but if either heat or moisture is excessive, the development of the virus is checked for a time.

(*c*) *Winds.*—Many facts are on record which seem to indicate that the poison may be carried to some distance by winds. The planting of trees has been held to interfere with the transmission by prevailing winds. Possibly, however, the quickly growing trees, such as the *Eucalyptus globulus*, have acted more beneficially by drying the soil.

(5) **Specific Gravity.**—That the distribution of the poison of malaria is influenced by gravity has long been conceded. Persons dwelling in the upper stories, or in buildings elevated some distance above the ground, are exempt in a marked degree.

The Specific Germ.—As Hirsch correctly remarks, the late J. K. Mitchell "was the first to approach in a scientific spirit the nature of infective disease and particularly in malarial fever." Many attempts were made to discover a constant and characteristic organism. Klebs and Tommasi-Crudeli in 1879 announced the discovery of a *bacillus malariæ*, but their observations have not been confirmed. In 1880 Laveran, a French army surgeon, now professor at the Medical School at Val de Grace, announced the discovery of a parasite in the blood of patients attacked by malarial fever. During the next three years he published nine additional communications, but for a time these observations attracted little attention. The Italian observers Marchiafava, Celli, and Golgi corroborated Laveran's statements. Councilman carefully studied the question in this country, and Laveran's statements were confirmed by myself in Philadelphia, by Walter James in New York, and more recently by Dock in Galveston. In India, Vandyke Carter has published an elaborate monograph on the parasites. In France, Germany, and England, owing in great part to the absence of cases of malaria, the value of Laveran's observations has been overlooked, but recently the confirmation has been published from many of the German clinics. So far as I know, not a single observer, who has had the necessary training and the material at his command, has failed to demonstrate the existence of these parasites.

The bodies which have been found invariably associated with all forms of malarial fevers, belong to the protozoa and to a group of organisms known as the *hæmatozoa*, the precise affinities of which have not yet been definitely determined. In some respects they closely resemble the monads,

in others the sporozoa. Parasites of the red blood-corpuscles have been met with abundantly in the blood of fish, turtles, and many species of birds. One of the best and most readily studied examples is the *Drepanidium ranarum*, a common parasite in the red blood-corpuscles of the frog.* In the blood of patients with malarial fevers the following forms may be seen: (1) an unpigmented hyaline body within the red blood-corpuscles which displays active movements; (2) a pigmented amœboid body within the red blood-corpuscles, which, under certain circumstances, may increase in size and form (3) a segmenting body, in which the protoplasm divides into a variable number of definite small spheres; (4) crescentic bodies, the so-called *crescents*, which develop within the blood-corpuscles and form characteristic and distinctive structures; (5) flagellate organisms, which may be seen to develop from the intercellular pigmented forms, or from ovoid bodies which are altered crescents; (6) free flagella. To the amœboid from within the red blood-corpuscles Marchiafava and Celli gave the name *plasmodium malariæ*. The following statements may be made with reference to these bodies:

The highest living authorities on protozoa, such as Bütschli, of Heidelberg, acknowledge that they are truly parasitic organisms. The testimony is now unanimous in France, India, America, Italy, and Germany that these bodies are always present in the malarial fevers. There is no evidence to show that they are ever present in any other disease. I can speak on this point with some confidence, having for years been in the habit of making blood examinations.

The relation of the parasites to the symptoms of the disease has been worked out in part by Golgi, who has shown that corresponding to the paroxysm there is a process of segmentation.

The relation of the different phases of growth to the varieties of malarial fever has not yet been thoroughly established, but the following points may be referred to: The typical intermittents are associated with large forms of the parasites, of which several varieties have been described. Golgi has described two distinct forms which he considers the causes of tertian and quartan fevers, and makes all other types depend on combinations of these. This probably holds good for a large proportion of intermittents. With the remittents, Marchiafava and Celli have described a distinct species, and look upon the crescents as representing a phase in its development. The pernicious malarial fevers are also associated with this variety, which the Italian observers call the "small plasmodium." The crescents may occur also in acute cases, but are most constant in malarial cachexia. The flagellate bodies do not appear to have any definite relation to the different forms of the disease.

The general symptoms and the morbid anatomy of malaria are in har-

* For an excellent account of these hæmatozoa and their development, see *Celli*, in Fortschritte der Medicin, 1891.

mony with the changes which this parasite induces. The destruction of the red blood-corpuscles by it can be traced in all stages. The presence of the pigment in the blood and the viscera, so characteristic of malaria, results from the transformation of the hæmoglobin by the plasmodia. The anæmia is a direct consequence of the wide-spread destruction of the corpuscles by the parasites. The constancy of their presence, the fact of their causing rapid destruction of the red blood-corpuscles, and the remarkable coincidence of their disappearance contemporaneously with the symptoms on the administration of quinine, are points strongly in favor of their etiological relation with the disease. There are still many gaps in our knowledge. We do not know how the parasite enters, or how or in what form it leaves the body; how and where it is propagated; under what outside conditions it develops, whether free or in some aquatic plant or animal. No record of its successful cultivation has been published.

Meantime, awaiting further knowledge, advantage may be taken of its constant presence in malaria. This alone, without reference to the true nature of the organism, is a fact of the highest importance. To be able, everywhere and under all circumstances, to differentiate between malaria and other forms of fever is one of the most important advances which has been made of late years in practical medicine, one which will revolutionize the study of fevers in tropical and subtropical countries, and should, within a short time, bring some order out of the chaos which at present exists regarding the different forms which there prevail.*

Morbid Anatomy.—The changes result from the disintegration of the red blood-corpuscles, accumulation of the pigment thereby formed, and possibly the influence of toxic materials produced by the parasite. Cases of simple malarial infection, the ague, are rarely fatal, and our knowledge of the morbid anatomy of the disease is drawn from the pernicious malaria or the chronic cachexia. Rupture of the enlarged spleen may occur spontaneously, but more commonly from trauma. A case of the kind was admitted under my colleague, Halsted, in June, 1889, and Dock has recently reported two cases.

(1) **Pernicious Malaria.**—The condition depends upon the duration of the infection and upon whether the patient has had previous attacks. The blood is hydræmic and the serum may even be tinged with hæmoglobin. The red blood-corpuscles present the endoglobular forms of the parasite and are in all stages of destruction. The spleen is enlarged, often only moderately; thus, of two fatal cases recently in my wards the spleens measured 13 × 8 ctm. and 14 × 8 ctm. respectively. If a fresh

* One rises from the perusal of the recent *Traité des Maladies des Pays Chauds*, by Kelsch and Kiener, with a feeling that the key to many of the complex problems there discussed and a totally different conception of many of the features of malaria would have been obtained had they studied the disease from the standpoint of their countryman Laveran.

infection, the spleen is usually very soft, and the pulp lake-colored and turbid. In cases of intense reinfection the spleen may be enlarged and firm. The amount of pigment in the spleen elements is enormously increased. The liver is swollen and turbid. In very acute cases there is not necessarily any macroscopic pigmentation, though microscopically the capillaries may be stuffed with degenerating red blood-corpuscles deeply pigmented. Perivascular (portal) infiltration has been found in a very acute case in a young man (Dock). The brain usually shows interesting changes. In severe cases of some duration the tissue is stained, sometimes chocolate-colored. In mild cases the discoloration is present, but less marked. The blood-vessels, especially the arterioles and capillaries, contain large numbers of parasites, with partial or total destruction of red blood-corpuscles, and pigmented leucocytes. Occlusions of arterioles by means of parasites are often seen. Anæmia and œdema are commoner than congestion. The kidneys show analogous conditions.

(2) **Malarial Cachexia.**—A patient, the subject of chronic paludism, usually dies of anæmia or of hæmorrhage associated with it. The most characteristic cases of the kind which have come under my observation have been in the workmen returning from the Panama Canal, victims of the so-called Chagres fever

The anæmia is profound, particularly if the patient has died of fever. The spleen is greatly enlarged, and may weigh from seven to ten pounds. If the disease has persisted for any length of time, it is firm and resists cutting. The capsule is thickened, the parenchyma brownish or yellowish-brown, with areas of pigmentation, or in very protracted cases it is extremely melanosed, particularly in the trabeculæ and about the vessels.

The liver may be greatly enlarged; but, as a rule, the increase in size is moderate in proportion to that of the spleen. It may present to the naked eye a grayish-brown or slate color due to the large amount of pigment. In the portal canals and beneath the capsule the connective tissue is impregnated with melanin. Varying with the duration of the disease, the shade of color of the liver ranges from a light gray to a deep slate-gray tint. The texture is firm, but there is not necessarily any great increase in the connective tissue. Histologically, the pigment is seen in the Kupffer's cells and the perivascular tissue.

The kidneys may be enlarged and present a grayish-red color, or areas of pigmentation may be seen. The pigment may be diffusely scattered and particularly marked about the blood-vessels and the Malpighian bodies, or it is often abundant in the cells of the convoluted and collecting tubules. The peritonæum is usually of a deep slate-color. The mucous membrane of the stomach and intestines may have the same hue, due to the pigment in and about the blood-vessels. In some cases this is confined to the lymph nodules of Peyer's patches, causing the shaven-beard appearance.

(3) The Accidental and Late Lesions of Malarial Fever.

(*a*) *The Liver.*—Paludal hepatitis plays a very important *rôle* in the history of malaria, as described by French writers. Kelsch and Kiener devote over sixty pages to a description of the various forms, parenchymatous and interstitial, describing under the latter three different varieties. The perusal of this section of their work by no means carries conviction that all the forms which they describe are associated definitely with malaria. Many of the patients were the subjects of chronic alcoholism, and the most important diagnostic point upon which they seem to have placed reliance was melanosis of the spleen, sometimes with pigmentation of Glisson's sheath. The existence of a cirrhosis dependent upon the irritation of large quantities of pigment in the liver is unquestioned, but only those cases in which the history of chronic malaria is definite, and in which the melanosis of both liver and spleen coexist, should be regarded as of paludal origin. The affection in this country is of extraordinary rarity. In the post-mortem room of the Philadelphia Hospital I have frequently seen, in subjects in whom the spleen was deeply pigmented, the portal sheaths of the liver stained, and a slight increase in the connective tissue; but it is begging the question to say that in such patients, who have almost certainly been habitual consumers of bad whisky, the condition of the liver was due to malaria. No instance of malarial cirrhosis has been shown at the Philadelphia Pathological Society since its foundation. Welch tells me he knows of but one specimen which has been shown in New York, and that was from an Algerian.

(*b*) *Pneumonia* is believed by many authors to be common in malaria, and even to depend directly upon the malarial poison, occurring either in the acute or in the chronic forms of the disease. I have no personal knowledge of such a special pneumonia It certainly does not occur in the intermittent or remittent fevers which prevail in Philadelphia and Baltimore. The question was formerly warmly discussed in this country, and I may refer to the attempt on the part of Manson to distinguish special forms depending upon the malarial poison. The exhaustive and critical review of the subject by W. T. Howard, in 1859, put a check to many of the speculations on the subject. The French authors quoted above recognize as common in chronic paludism a form distinguished by an irregular course, an absence of many of the characteristic symptoms, by a rapid swelling of the liver and spleen, and a special tendency to the formation of necrotic foci. On two occasions in the Philadelphia Hospital I had an opportunity of seeing the development of pneumonia in convalescents from malaria—one of a quotidian, the other of a quartan type. They developed in a ward with several other cases of pneumonia, and the disease ran a perfectly normal course. In about four hundred cases of malaria which have been under observation at the Johns Hopkins Hospital and Dispensary bronchitis has been frequent as an early symptom,

but we have seen no indication of any special form of inflammation of the lungs.

(*c*) *Nephritis.*—Acute inflammation of the kidneys is rare in the milder forms. Albumen in the urine is not infrequent during the chill, and in the course of the continued or remittent fevers. Kelsch and Kiener describe several forms of nephritis. No instance of acute or chronic Bright's disease resulting directly from paludism has come under my notice.

Clinical Forms of Malarial Fever.—(1) **Intermittent Fever.**—This form is characterized by recurring paroxysms of what are known as ague, in which, as a rule, chill, fever, and sweat follow each other in orderly sequence. The stage of *incubation* may be very short. Attacks have occurred within twenty-four hours after exposure. Usually the time of incubation is from seven to fourteen days. On the other hand, the ague may be, as is said, "in the system," and the patient may have a paroxysm months after he has removed from a malarial region, though I doubt if this can be the case unless he has had the disease when living there.

Description of the Paroxysm.—The patient generally knows he is going to have a chill a few hours before its advent by unpleasant feelings and uneasy sensations, sometimes by headache. The paroxysm is divided into three stages—cold, heat, and sweating.

Cold Stage.—The onset is indicated by a feeling of lassitude and a desire to yawn and stretch, by headache, uneasy sensations in the epigastrium, sometimes by nausea and vomiting. Even before the chill begins the thermometer indicates slight rise in temperature. Gradually the patient begins to shiver, the face looks cold, and in the fully developed rigor the whole body shakes, the teeth chatter, and the movements may often be violent enough to shake the bed. Not only does the patient look cold and blue, but a surface thermometer will indicate a reduction of the skin temperature. On the other hand, the axillary or rectal temperature may, during the chill, be greatly increased, and, as shown in the chart, the fever may rise during the chill to 105° or 106°. Of symptoms associated with the chill, nausea and vomiting are common. There may be intense headache. The pulse is quick, small, and hard. The urine is increased in quantity. The chill lasts for a variable time, from ten or twelve minutes to an hour, or even longer.

The *hot stage* is ushered in by transient flushes of heat; gradually the coldness of the surface disappears and the skin becomes intensely hot. The contrast in the patient's appearance is striking: the face is flushed, the hands are congested, the skin reddened, the pulse is full and bounding, the heart's action is forcible, and the patient may complain of a throbbing headache. The rectal temperature may not increase much during this stage; in fact, by the termination of the chill the fever may have reached its maximum. The duration of the hot stage varies from half an

No. 779 Clarence Jones ADMITTED Jan. 30th 1890 WARD

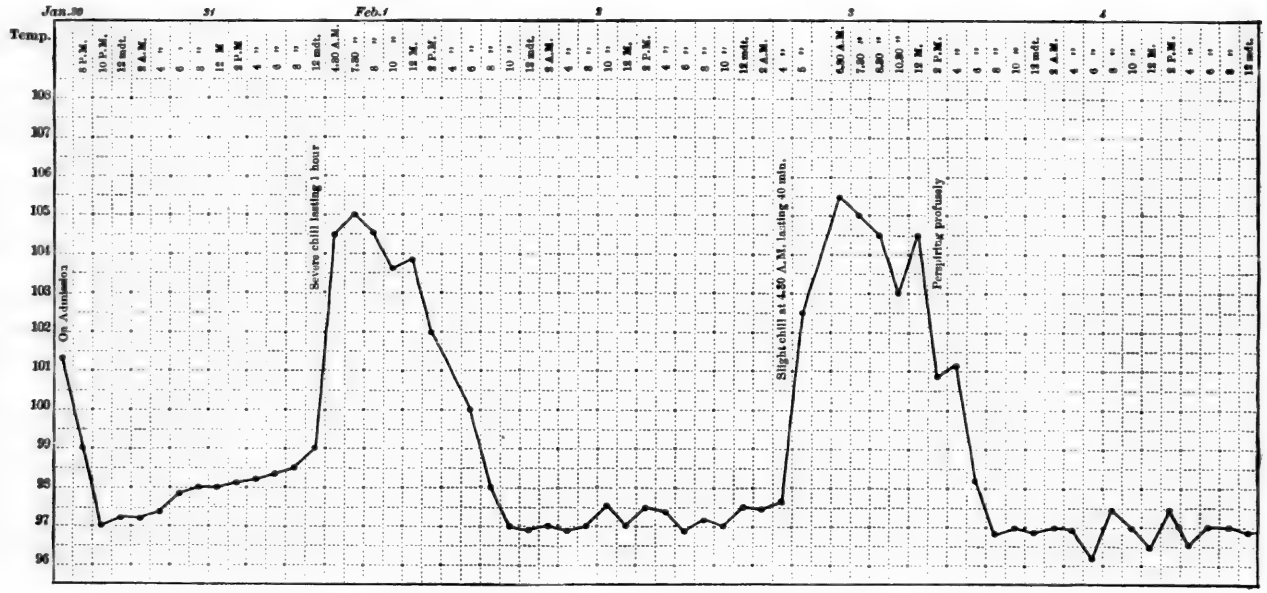
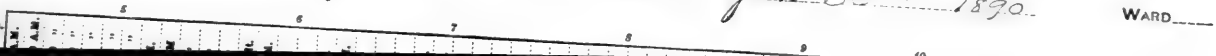


CHART XI.

No. 779 Clarence Jones ADMITTED Jan 30th 1890 WARD



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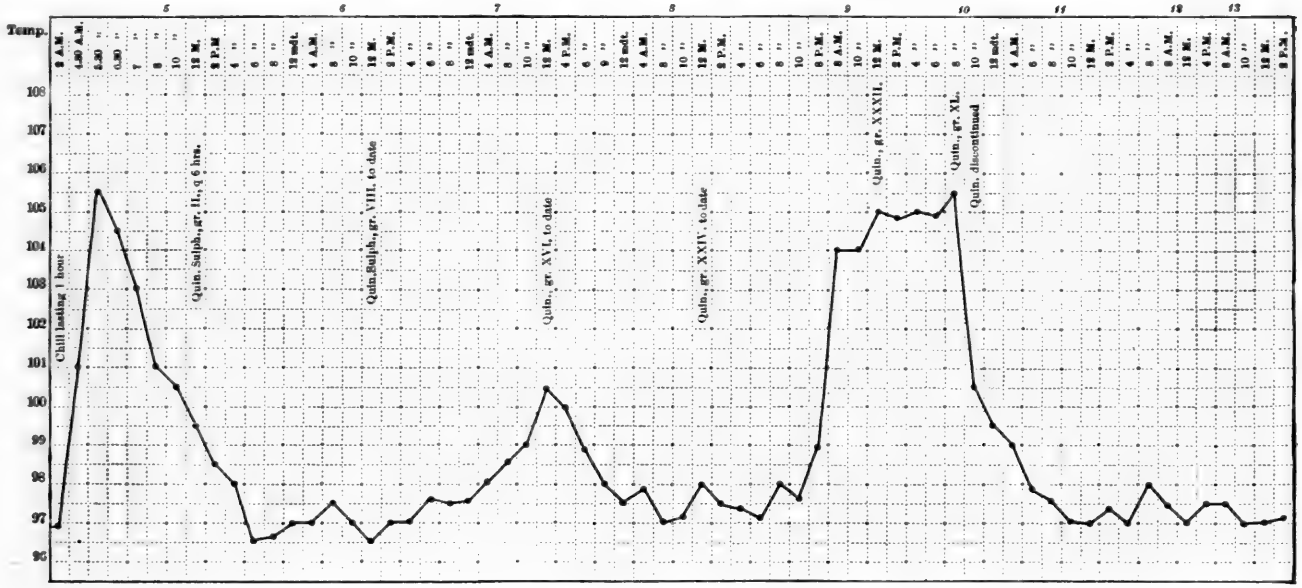


CHART XI.—*(Continued).*

hour to three or four hours. The patient is intensely thirsty and drinks eagerly of cold water.

Sweating Stage.—Beads of perspiration appear upon the face and gradually the entire body is bathed in a copious sweat. The uncomfortable feeling associated with the fever disappears, the headache is relieved, and within an hour or two the paroxysm is over and the patient usually sinks into a refreshing sleep. The sweating varies much. It may be drenching in character or it may be slight.

Chart XI is a fac-simile of a ward temperature chart in a case of tertian ague. The duration of the paroxysms on February 1st, 3d, and 5th was from twelve to sixteen hours. Quinine in two-grain doses was given on the 5th and was sufficient to prevent the on-coming paroxysms on the 7th, though the temperature rose to 100·5°. The small doses, however, were not effective, and on the 9th he had a severe chill.

The total duration of the paroxysm is from twelve to fifteen hours, but may be shorter. Variations in the paroxysm are common. Thus the patient may, instead of a chill, experience only a slight feeling of coldness. The most common variation is the occurrence of a hot stage alone, or with very slight sweating. During the paroxysm the spleen is enlarged and the edge can usually be felt below the costal margin. In the interval or intermission of the paroxysm the patient feels very well, and, unless the disease is unusually severe, he is able to be up. Bronchitis is a common symptom. Herpes, usually labial, is perhaps as frequently seen in ague as in pneumonia.

Types of the Paroxysm.—The periodicity of the paroxysms is one of the most striking features in malarial fever. They occur with regularity, either at the end of twenty-four. forty-eight, or seventy-two hours.

Returning at the end of twenty-four hours the paroxysm is daily, hence the name *quotidian*. This is by far the most frequent type in the acute intermittent fevers in this latitude. Should two attacks occur daily, which is very rare, it is called a double quotidian. The observations of the Italian observers, more particularly Golgi, have enabled us to trace certain definite cycles of evolution in the development of the malarial parasites, and in the character of the organism in the different forms of the disease. In the quotidian type the plasmodia are small at first and display active movements. The parasite gradually increases in size, fills the entire corpuscle, or at the beginning and prior to the paroxysm undergoes in many of the corpuscles segmentation or sporulation.

If the paroxysm occurs at the end of forty-eight hours, it happens upon the third day; hence the term *tertian* applied to this form. This is the next most frequent form, but it is much less common than the quotidian form. In the tertian type the blood-corpuscles contain small amœboid bodies which gradually develop, become deeply pigmented, and, according to Golgi, the segmentation consists of from fifteen to twenty separate

bodies, aggregated about the central clump of pigment. Here, too, the segmentation occurs just prior to and during the chill.

Occurring at the end of seventy-two hours the paroxysm is on the fourth day, hence the name *quartan* ague. This is rare, and in the past seven years I have met with but six or eight well-marked instances. In this form the amœboid bodies become rapidly pigmented. The movements are slower. They grow gradually in the red blood-corpuscles, and, according to Golgi, attain considerable size without removing all the hæmoglobin from the corpuscles. Prior to the chill, segmentation of the bodies takes place into from nine to twelve different portions.

Other types, such as quintan or sextan, need not be considered, as they are very rare.

Course of the Disease.—After a few paroxysms, or after the disease has persisted for ten days or two weeks, the patient may get well without any special medication. In cases in which we have been studying the hæmatozoa I have repeatedly known the chills to stop spontaneously. Such cases, however, are very liable to recurrence. Persistence of the fever leads to anæmia and a hæmatogenous jaundice, owing to the destruction of the red blood-disks by the parasites. Ultimately the condition may become chronic, and will be described under malarial cachexia. Cases of intermittent fever yield promptly and immediately to treatment by quinine.

(2) **Continued and Remittent Form of Malarial Fever.**—Under this head will be described that form of fever in which there are no distinct intermissions, but in which the temperature range is constantly above normal, though there are marked remissions. It is not an uncommon disease in this locality. The severer forms of it prevail in the Southern States and in tropical countries where it is known chiefly as *bilious remittent fever*. The entire group of cases included under the terms *remittent fever*, *bilious remittent*, and *typho-malarial fevers* requires to be studied anew in the light of Laveran's observations.

Symptoms.—The disease may set in with a definite chill, or may be preceded for a few days by feelings of *malaise*. As seen in this latitude, the patient has either chilliness or a distinct rigor in the beginning. When seen on the second or third day of the disease he has a flushed face and looks ill. The tongue is furred, the pulse is full and bounding, but rarely dicrotic. The temperature may range from 102° to 103°, or is in some instances higher. The general appearance of the patient is strongly suggestive of typhoid fever, a suggestion still further borne out by the existence of acute splenic enlargement of moderate grade. As in intermittent fever, an initial bronchitis may be present. The course of these cases is variable. The fever is continuous, with remissions more or less marked; definite paroxysms with or without chills may occur, in which the temperature rises to 105° or 106°. Intestinal symptoms are not present. A slight hæmatogenous jaundice may develop early. Delirium, usually of a

mild type, may occur. The cases vary greatly in severity. In some the fever subsides at the end of the week, and the practitioner is in doubt whether he has had to do with a mild typhoid or a simple febricula. In other instances the fever persists for from ten days to two weeks, there are marked remissions, perhaps chills, with a furred tongue and low delirium. Jaundice is not infrequent. These are the cases to which the term bilious remittent and typho-malarial fevers are applied. In other instances the symptoms become grave and assume a character of the pernicious type. It is this form of malarial fever about which so much confusion still exists. The similarity of the cases at the outset to typhoid fever is most striking, more particularly the appearance of the facies, and the patient *looks* very ill. The cases develop, too, in the autumn, at the very time when typhoid fever occurs. The fever yields, as a rule, promptly to quinine, though here and there cases are met with, rarely indeed in my experience, in which they are refractory. It is just in this group that the observations of Laveran will be found of the greatest value.

The *diagnosis* of malarial remittent fever may be definitely made by the examination of the blood. Vandyke Carter, in his monograph, alludes to the value of this method in the fevers of India. In many cases here we are at first unable to distinguish between typhoid and continued malarial fever without a blood examination. A more wide-spread use of this means of diagnosis will enable us to bring some order out of the confusion which exists in the classification of the fevers of the South. At present the following febrile affections are recognized by various physicians as occurring in the subtropical regions of this continent: (*a*) Typhoid fever; (*b*) typho-malarial fever, a typhoid modified by malarial infection, or the result of a combined infection; (*c*) the malarial remittent fever; and (*d*) continued thermic fever (Guitéras). In these various forms, all of which may be characterized by a continued pyrexia with remissions or with chills and sweats (for we must remember that chills and sweats in typhoid fever are by no means rare), the blood examination will enable us to discover those which depend upon the malarial poison. In this latitude we have not the opportunity of seeing many of the protracted and severe cases, but I am inclined to think that future observations will show that apart from the thermic fever there are only two forms of these continued fevers in the South—the one due to the *typhoid*, and the other to the *malarial* infection. The typhoid fever of Philadelphia and Baltimore presents no essential difference from the disease as it occurs in Montreal, a city practically free from malaria. Dock has shown conclusively that cases diagnosed in Texas as continued malarial fever were really true typhoid.

(3) **Pernicious Malarial Fever.**—This is fortunately rare in temperate climates, and the number of cases which now occur, for example, in Philadelphia and Baltimore, is very much less than thirty or forty years ago. Among the cases of malaria which have been under observation during

the past two years there were only two of the pernicious form. The following are the most important types:

(*a*) The *comatose form*, in which a patient is struck down with symptoms of the most intense cerebral disturbance, either acute delirium or, more frequently, a rapidly developing coma. A chill may or may not precede the attack. The fever is usually high, and the skin hot and dry. The unconsciousness may persist for from twelve to twenty-four hours, or the patient may sink and die. After regaining consciousness a second attack may come on and prove fatal.

(*b*) *Algid Form.*—In this, the attack sets in usually with gastric symptoms; there are vomiting, intense prostration, and feebleness out of all proportion to the local symptoms. The patient complains of feeling cold, although there may be no actual chill. The temperature may be normal or even subnormal; consciousness may be retained. The pulse is feeble and small, and the respirations are increased. The urine is often diminished or even suppressed. This condition may persist with slight exacerbations of fever for several days and the patient may die in a condition of profound asthenia. In a recent case the patient, admitted on October 10th, had been ill since the 7th, but there were no chills. When first seen he was prostrated and weak, and looked as if he had been drinking, but there was no alcoholic odor of the breath, and on learning that he had recently come from Savannah, the blood was at once examined and large numbers of Laveran's organisms were found, chiefly of the small intracorpuscular variety. The temperature was only 101°. During the next five days the prostration, extreme depression, and vomiting continued; the pulse ranged from 70 to 80, and the temperature, after the first day, did not rise above 98°, but sank as low as 96°. This is essentially the same as described as the *asthenic* or *adynamic* form of the disease.

(*c*) *Hæmorrhagic Forms.*—In all the severe types of malarial infection, especially if persistent, hæmorrhage may occur from the mucous membranes. An important form is the malarial *hæmaturia*, which in some instances assumes a very malignant type. Paroxysms of ague may precede the attack, but in many cases called malarial hæmaturia there is no febrile paroxysm. The condition is usually hæmoglobinuria, though blood-corpuscles are present also. In severe cases there is bleeding from the mucous membranes. Jaundice is present, but to a variable extent, and is hæmatogenous, due to the destruction of the red blood-corpuscles. Malarial hæmaturia occurs in epidemic form in many regions of the Southern States, and in some seasons proves very fatal.

Many different forms of pernicious malarial fever—diaphoretic, syncopal, pneumonic, pleuritic, choleraic, cardiac, gastric, and gangrenous—all of which depend upon some special symptom, have been described.

(4) **Malarial Cachexia.**—The symptoms of chronic malarial poisoning are very varied. It may follow the frequent recurrence of ordinary intermittent fever, a common sequence in this country. A patient has chills

for several weeks, is improperly or imperfectly treated, and on exposure the chills recur. This may be repeated for several months until the patient presents the two striking features of malarial cachexia—namely, *anæmia* and an *enlarged spleen*. Cases developing without chills or without febrile paroxysms are almost unknown in this region. They may occur, however, in intensely malarial districts, but in such cases the patients have fever, though chills may not supervene. The most pronounced types of malarial cachexia which we meet with here are in sailors from the West Indies and Central America. There is profound anæmia; the blood count may be as low as one million per cubic millimetre; the skin has a saffron-yellow or lemon tint, not often the light-yellow tint of pernicious anæmia, but a darker, dirtier yellow. The spleen is greatly enlarged, firm, and hard. It rarely reaches the dimensions of the large leukæmic organ, but comes next to it in size.

The general symptoms are those of ordinary anæmia—breathlessness on exertion, œdema of the ankles, hæmorrhages, particularly into the retina, as noted by Stephen Mackenzie. Occasionally the bleeding is severe, and I have twice known fatal hæmatemesis to occur in association with the enlarged spleen. The fever is variable. The temperature may be low for days, not reaching above 99·5°. In other instances there may be irregular fever, and the temperature rises gradually to 102·5° to 103°. The cases in fact present a picture of splenic anæmia.

With careful treatment the outlook is good, and a majority of cases recover. The spleen is gradually reduced in size, but it may take several months or, indeed, in some instances, several years before the ague-cake entirely disappears.

Among the rarer symptoms which may develop as a result of malarial intoxication may be mentioned *paraplegia*, cases of which have been described by Gibney, Suckling, and others. Some of the cases are doubtful, and have been attributed to malaria simply because the paralysis was intermittent. It is a condition of extreme rarity. No case is mentioned by Kelsch and Kiener. Suckling's case had had several attacks of malaria, the last of which preceded by about two weeks the onset of the nervous symptoms, which were headache, giddiness, loss of speech, and paraplegia. The attack was transient, but he had a subsequent attack which also followed an ague-fit. The patient was an old soldier who had had syphilis, a point which somewhat complicated the case. *Orchitis* has been described as developing in malaria by Charvot in Algiers and Fedeli in Rome.

Diagnosis.—The diagnosis of the various forms of malaria is usually easy. The continued and remittent and certain of the pernicious cases offer difficulties, which, however, are now greatly lessened or entirely overcome since Laveran's researches have given us a positive diagnostic indication. Many forms of intermittent pyrexia are mistaken for malarial fever, particularly the initial chills of tuberculosis and of septic infection.

If the practitioner will take to heart the lesson that an intermittent fever which resists quinine is not malarial, he will avoid many errors in diagnosis. In the so-called masked intermittent or dumb ague, the febrile manifestations are more irregular and the symptoms less pronounced; but occasionally chills occur, and the therapeutical test usually removes every doubt in the diagnosis.

The malarial poison is supposed to influence many affections in a remarkable way, giving to them a paroxysmal character. A whole series of minor ailments and some more severe ones, such as neuralgia, are attributed to certain occult effects of paludism. The more closely such cases are investigated the less definite appears the connection with malaria. Practitioners in districts entirely exempt from the disease have to deal with ailments which present the same odd periodicity, and which the physicians of the Atlantic coast attribute to a "touch of malaria."

Treatment.—We do not know as yet how the poison reaches the system. Infection seems most liable to occur at night. In regions in which the disease prevails extensively the drinking-water should be boiled. Persons going to a malarial region should take about ten grains of quinine daily. During the paroxysm the patient should, in the cold stage, be wrapped in blankets and given hot drinks. The reactionary fever is rarely dangerous even if it reaches a high grade. The body may, however, be sponged. Quinine should then be ordered, so as to check the on-coming paroxysm. It should be given in solution. From ten to thirty grains in divided doses through the day will almost invariably stop the next paroxysm. No preparatory treatment is necessary; no other drugs need be given. The remedy is a specific in the truest sense of the term. In not a single instance among the several hundred cases of intermittent fever which I have had under observation during the past seven years did quinine fail to check the paroxysms. The mode of administration is of little moment, so long as the patient gets a sufficient quantity into his system. In solution or in capsule it is the most efficient. The pills and compressed tablets are more uncertain, as they may not be dissolved. A question of interest is the efficient dose of quinine necessary to cure the disease. I have a number of charts showing that grain doses three times a day will, in many cases, prevent the paroxysm, but not always with the certainty of the larger doses. It is safer to give at least from twenty to thirty grains daily for the first three days and then to continue the remedy in smaller doses for two or three weeks. Other remedies in acute forms of malaria are useless.

In the pernicious forms, and when it is desirable to get the system as rapidly under its influence as possible, the drug should be administered hypodermically (as the bisulphate in thirty-grain doses with five grains of tartaric acid) every two or three hours. For the extreme restlessness in these cases opium is indicated, and cardiac stimulants (such as alcohol and strychnine) are necessary. If in the comatose form the internal tem-

perature is raised, the patient should be put in a bath and douched with cold water.

For malarial anæmia, iron and arsenic are indicated.

XXII. ANTHRAX.

(*Malignant Pustule; Splenic Fever; Charbon; Wool-sorter's Disease.*)

Definition.—An acute infectious disease caused by the *bacillus anthracis*. It is a wide-spread affection in animals, particularly in sheep and cattle. In man it occurs sporadically or as a result of accidental absorption of the virus.

Etiology.—The infectious agent is a non-motile, rod-shaped organism, the *bacillus anthracis*, which has, by the researches of Pollender, Davaine, Koch, and Pasteur, become the best known perhaps of all pathogenic microbes. The bacillus has a length of from two to ten times the diameter of a red blood-corpuscle; the rods are often united. They multiply by fission with great rapidity and grow with facility on various culture media, extending into long filaments which interlace and produce a dense mycelium. The spore formation is seen with great readiness in these filaments. The bacilli themselves are readily destroyed, but the spores are very resistant, and survive after prolonged immersion in a five-per-cent solution of carbolic acid, and resist for some minutes a temperature of 212° Fahr. They are capable also of resisting gastric digestion. Outside the body the spores are in all probability very durable.

Geographically and zoölogically the disease is the most wide-spread of all infectious disorders. It is much more prevalent in Europe and in Asia than in America. The ravages among the herds of cattle in Russia and Siberia, and among sheep in certain parts of Europe, are not equalled by any other animal plague. In this country the disease is rare. So far as I know it has never prevailed on the ranches in the Northwest, but cases were not infrequent about Montreal.

A protective inoculation with a mitigated virus has been introduced by Pasteur, and has been adopted in certain anthrax regions. Hankin has isolated from the cultures an albumose which renders animals immune against the most intense virus.

In animals the disease is conveyed sometimes by direct inoculation, as by the bites and stings of insects, by feeding on carcasses of animals which have died of the disease, but more commonly by feeding in pastures in which the germs have been preserved. Pasteur believes that the earthworm plays an important part in bringing to the surface and distributing the bacilli which have been propagated in the buried carcass of an infected animal. Certain fields, or even farms, may thus be infected for an indefinite period of time. It seems probable, however, that if the carcass

is not opened or the blood spilt, spores are not formed in the buried animal.

Animals vary in susceptibility: herbivora in the highest degree, then the omnivora, and lastly the carnivora. The disease does not occur spontaneously in man, but always results from infection, either through the skin, the intestines, or in rare instances through the lungs. The disease is found in persons whose occupations bring them into contact with animals or animal products, as stablemen, shepherds, tanners, butchers, and those who work in wool and hair.

Various forms of the disease have been described, and two chief groups may be recognized: the external anthrax, or malignant pustule, and the internal anthrax, of which there are pulmonary and intestinal forms.

Symptoms.—(1) External Anthrax.

(*a*) *Malignant Pustule.*—The inoculation is usually on an exposed surface—the hands, arms, or face. At the site of inoculation there are, within a few hours, itching and uneasiness. Gradually a small papule develops, which becomes vesicular. Inflammatory induration extends around this, and within thirty-six hours, at the site of inoculation there is a dark brownish eschar, at a little distance from which there may be a series of small vesicles. The brawny induration may be extreme. The œdema produces very great swelling of the parts. The inflammation extends along the lymphatics, and the neighboring lymph-glands are swollen and sore. The temperature at first rises rapidly, and the febrile phenomena are marked. Subsequently the fever falls, and in many cases becomes subnormal. Death may take place in from three to five days. In cases which recover the constitutional symptoms are slighter, the eschar gradually sloughs out, and the wound heals. The cases vary much in severity. In the mildest form there may be only slight swelling. At the site of inoculation a papule is formed, which rapidly becomes vesicular and dries into a scab, which separates in the course of a few days.

(*b*) *Malignant Anthrax Œdema.*—This form occurs in the eyelid, and also in the head, hand, and arm, and is characterized by the absence of the papule and vesicle forms, and by the most extensive œdema, which may follow rather than precede the constitutional symptoms. The œdema reaches such a grade of intensity that gangrene results, and may involve a considerable surface. The constitutional symptoms then become extremely grave, and the cases invariably prove fatal.

A feature in both these forms of malignant pustule, to which many writers refer, is the absence of feeling of distress or anxiety on the part of the patient, whose mental condition may be perfectly clear. He may be without any apprehension, even though his condition is very critical.

The *diagnosis* in most instances is readily made from the characters of the lesion and the occupation of the patient. When in doubt, the examination of the fluid from the pustule may show the presence of the anthrax bacilli. Cultures should be made, or a mouse or guinea-pig inocu-

lated. It is to be remembered that the blood may not show the bacilli in numbers until shortly before death.

(2) **Internal Anthrax.**

(*a*) *Intestinal Form, Mycosis intestinalis.*—In these cases the infection is through the stomach and intestines, and results from eating the flesh or drinking the milk of diseased animals. The symptoms are those of intense poisoning. The disease may set in with a chill, followed by vomiting, diarrhœa, moderate fever, and pains in the legs and back. In acute cases there are dyspnœa, cyanosis, great anxiety and restlessness, and toward the end convulsions or spasms of the muscles. Hæmorrhage may occur from the mucous membranes. Occasionally there are small phlegmonous areas on the skin, or petechiæ develop. The spleen is enlarged. The blood is dark and remains fluid for a long time after death. Late in the disease the bacilli may be found in the blood.

This is one of the forms of acute poisoning which may affect many individuals together. Thus Butler and Karl Huber describe an epidemic in which twenty-five persons were attacked after eating the flesh of an animal which had had anthrax. Six died in from forty-eight hours to seven days.

(*b*) *Wool-sorter's Disease.*—This important form of anthrax is found in the large establishments in which wool or hair is sorted and cleansed. The hair and wool imported into Europe from Russia and South America appear to have induced the largest number of cases. Many of these cases show no external lesion. The infection has been swallowed or inhaled with the dust. There are rarely premonitory symptoms. The patient is seized with a chill, becomes faint and prostrated, has pains in the back and legs, and the temperature rises to 102° to 103°. The breathing is rapid, and he complains of much pain in the chest. There may be a cough and signs of bronchitis. So prominent in some instances are these bronchial symptoms that a pulmonary form of the disease has been described. The pulse is feeble and very rapid. There may be vomiting, and death may occur within twenty-four hours with symptoms of profound collapse and prostration. Other cases are more protracted, and there may be diarrhœa, delirium, and unconsciousness. The recognition of wool-sorter's disease as a form of anthrax is due to J. H. Bell, of Bradford, England.

In certain instances these profound constitutional symptoms of internal anthrax are associated with the external lesions of malignant pustule.

The diagnosis of internal anthrax is by no means easy, unless the history points definitely to infection in the occupation of the individual. In cases of doubt cultures should be made, and inoculations performed in animals. Some of these cases may possibly be caused by organisms other than the bacillus of anthrax (Cornil and Babes).

Treatment.—In malignant pustule the site of inoculation should be destroyed by the caustic or hot iron, and powdered bichloride of mercury

may be sprinkled over the exposed surface. The local development of the bacilli about the site of inoculation may be prevented by the subcutaneous injections of solutions of carbolic acid or bichloride of mercury. The injections should be made at various points around the pustule, and may be repeated two or three times a day. The internal treatment should be confined to the administration of stimulants and plenty of nutritious food. Davies-Colley advises ipecacuanha powder in doses of from five to ten grains every three or four hours.

In malignant forms, particularly the intestinal cases, little can be done. Active purgatives may be given at the outset, so as to remove the infecting material. Quinine in large doses has been recommended.

XXIII. RABIES.

(*Lyssa; Hydrophobia.*)

Definition.—An acute disease of animals, dependent upon a specific virus, and communicated by inoculation to man.

Etiology.—In man the disease is very variously distributed. In Russia it is common, in North Germany it is extremely rare, owing to the wise provision that all dogs shall be muzzled. In England and France it is much more common. In this country the disease is very rare. Since 1867 I have seen but two cases.

Canines are specially liable to the disease. It is found most frequently in the dog, the wolf, and the cat. All animals are, however, susceptible; and it is communicable by inoculation to the ox, horse, or pig. The disease is propagated chiefly by the dog, which seems specially susceptible. In the Western States the skunk is said to be very liable to the disease. The nature of the poison is as yet unknown. It is contained chiefly in the nervous system and is met with in the secretions, particularly in the saliva.

A variable time elapses between the introduction of the virus and the appearance of the symptoms. Horsley states that this depends upon the following factors: "(*a*) Age. The incubation is shorter in children than in adults. For obvious reasons the former are more frequently attacked. (*b*) Part infected. The rapidity of onset of the symptoms is greatly determined by the part of the body which may happen to have been bitten. Wounds about the face and head are especially dangerous: next in order in degrees of mortality come bites on the hands, then injuries on the other parts of the body. This relative order is, no doubt, greatly dependent upon the fact that the face, head, and hands are usually naked, while the other parts are clothed. (*c*) The extent and severity of the wound. Puncture wounds are the most dangerous; the lacerations are fatal in proportion to the extent of the surface afforded for absorption of

the virus. (*d*) The animal conveying the infection. In order of decreasing severity come: first, the wolf; second, the cat; third, the dog; and fourth, other animals." Only a limited number of those bitten by rabid dogs become affected by the disease; according to Horsley, not more than fifteen per cent. On the other hand, the death-rate of those persons bitten by wolves is higher, not less than forty per cent.

The incubation period in man is extremely variable. The average is from six weeks to two months. In a few cases it has been under two weeks. It may be prolonged to three months. It is stated that the incubation may be prolonged for a year or even two years, but this has not been definitely settled.

Symptoms.—Three stages of the disease are recognized:

(1) *Premonitory stage*, in which there may be irritation about the bite, or pain or numbness. The patient is depressed and melancholy; and complains of headache and loss of appetite. He is very irritable and sleepless, and has a constant sense of impending danger. There is often greatly increased sensibility. A bright light or a loud voice is distressing. The larynx may be injected and the first symptoms of difficulty in swallowing are experienced. The voice also becomes husky. There is a slight rise in the temperature and the pulse.

(2) *Furious Stage.*—This is characterized by great excitability and restlessness, and an extreme degree of hyperæsthesia. "Any afferent stimulant—i. e., a sound or a draught of air, or the mere association of a verbal suggestion—will cause a violent reflex spasm. In man this symptom constitutes the most distressing feature of the malady. The spasms, which affect particularly the muscles of the larynx and mouth, are exceedingly painful and are accompanied by an intense sense of dyspnœa, even when the glottis is widely opened or tracheotomy has been performed" (Horsley). Any attempt to take water is followed by an intensely painful spasm of the muscles of the larynx and of the elevators of the hyoid bone. It is this which makes the patient dread the very sight of water and gives the popular name to the disease. These spasmodic attacks may be associated with maniacal symptoms. In the intervals between them the patient is quiet and the mind unclouded. The temperature in this stage is usually elevated and may reach from 100° to 103°. In some instances the disease is afebrile. The patient rarely attempts to injure his attendants, and in the intense spasms may be particularly anxious to avoid hurting any one. There are, however, occasional fits of furious mania, and the patient may, in the contractions of the muscles of the larynx and pharynx, give utterance to odd sounds. This stage lasts from a day and a half to three days and gradually passes into the—

(3) *Paralytic Stage.*—In rodents the preliminary and furious stages are absent, as a rule, and the paralytic stage may be marked from the outset—the so-called dumb rabies. This stage rarely lasts longer than from six to eighteen hours. The patient then becomes quiet; the spasms no

longer occur; there is gradual unconsciousness; the heart's action becomes more and more enfeebled, and death occurs by syncope.

Morbid Anatomy.—The lesions are in the cerebro-spinal system. The blood-vessels are congested; there is perivascular exudation of leucocytes; and there are minute hæmorrhages. According to Gowers, these are particularly intense in the medulla. The pharynx is congested, the mucous membrane of the stomach is hyperæmic, and not infrequently covered with a blood-stained mucus. The larynx, trachea, and bronchi show acute congestion. There are no special changes in the abdominal or thoracic viscera. The inoculation experiments show that the virus is not present in the liver, spleen, or kidneys, but is abundant in the spinal cord and brain.

Treatment.—Prophylaxis is of the greatest importance, and by a systematic muzzling of dogs the disease can be, as in Germany, practically eradicated.

The bites should be carefully washed and thoroughly cauterized with caustic potash or concentrated carbolic acid. It is best to keep the wound constantly open for at least five or six weeks. When once established the disease is hopelessly incurable. No measures have been found of the slightest avail, consequently the treatment must be palliative. The patient should be kept in a darkened room, in charge of not more than two careful attendants. To allay the spasm, chloroform may be administered and morphia given hypodermically. It is best to use these powerful remedies from the outset, and not to temporize with chloral, bromide of potassium, and other less potent drugs. By the local application of cocaine, the sensitiveness of the throat may be diminished sufficiently to enable the patient to take liquid nourishment. Sometimes he can swallow readily. Nutrient enemata may be administered.

Preventive Inoculation.—Pasteur has found that the virus, when propagated through a series of rabbits, increases rapidly in its virulence; so that whereas subdural inoculation from the brain of a mad dog takes from fifteen to twenty days to produce the disease, in successive inoculations in a series of rabbits the incubation period is gradually reduced to seven days. The spinal cord of these rabbits contains the virus in great intensity, but when preserved in dry air the virus gradually diminishes in intensity. If now dogs are inoculated with cords preserved for from twelve to fifteen days, and then with cords preserved for a shorter period, i. e., with a progressively stronger virus, they gradually acquire immunity against the disease. A dog treated in this way will resist inoculation with material from a perfectly fresh cord from a rabid rabbit, which otherwise would inevitably have proved fatal. Relying upon these experiments, Pasteur began inoculations in the human subject using, on successive days, material from cords in which the virus was of varying degrees of intensity.

There is still much discussion as to the full value of this method, but if the protective inoculation can be successfully performed in dogs,

there is no reason why the same should not hold good for man; and the figures published annually from the Pasteur Institute show that in persons bitten by animals known to have been rabid, the mortality after inoculation is only about 0·60 per cent.

Pseudo-rabies.—This is a very interesting affection, which may closely resemble hydrophobia, but is really nothing more than a neurotic or hysterical manifestation. A nervous person bitten by a dog, either rabid or supposed to be rabid, develops within a few months, or even later, symptoms somewhat resembling the true disease. He is irritable and depressed. He constantly declares his condition to be serious and that he will inevitably become mad. He may have paroxysms in which he says he is unable to drink, grasps at his throat, and becomes emotional. The temperature is not elevated and the disease does not progress. It lasts much longer than the true rabies, and is amenable to treatment. It is not improbable that a majority of the cases of alleged recovery in this disease have been of this hysterical form. In a case which Burr reported from my clinic a few years ago the patient had paroxysmal attacks in which he could not swallow. He was greatly excited and alarmed at the sight of water and was extremely emotional. The attack lasted for a couple of weeks and yielded to treatment with powerful electrical currents.

XXIV. TETANUS.

(*Lockjaw.*)

Definition.—An infectious malady characterized by tonic spasms of the muscles with marked exacerbations. The virus is produced by a bacillus which occurs in earth and sometimes in putrefying fluids and manure.

Etiology.—It occurs as an idiopathic affection or follows trauma. It is frequent in some localities and has prevailed extensively in epidemic form among new-born children, when it is known as tetanus or trismus neonatorum. It is more common in hot than in temperate climates, and in the colored than in the Caucasian race. This is particularly the case with tetanus following confinement and in tetanus neonatorum. In certain of the West India Islands more than one half of the mortality among the negro children has been due to this cause. In a majority of the cases there is an injury which may be of the most trifling character. It is more common after punctured and contused than after incised wounds, and frequently follows those of the hands and feet. The disease usually appears within two weeks of the injury. In some military campaigns tetanus has prevailed extensively, but in others, as in the late civil war, the cases have been comparatively few. Idiopathic tetanus is rare in man, but it has sometimes followed exposure to cold or after sleeping on the damp ground.

The infectious nature of tetanus was suggested by its endemic occurrence and from the manner of its behavior in certain institutions. Veterinarians have long been of this belief, as cases are apt to occur together in horses in one stable. In the United States attention was early called to this feature by the prevalence of the disease in the eastern end of Long Island.

The Tetanus Bacillus.—The observations of Rosenbach, Nicolaïer, and Kitasato have demonstrated that there is in connection with the disease a specific organism which can be isolated and cultivated. The bacillus forms a slender rod with rounded ends and may grow into long threads. It is motile, grows at ordinary temperatures, and is anaërobic. With small quantities of the culture the disease may be transmitted to animals, which die with symptoms of tetanus. An extremely interesting fact is the separation by Brieger, from the cultures as well as from a subject dead of tetanus, of poisonous substances capable of producing the disease. Of these ptomaines one, *tetanin*, causes the characteristic symptoms of tetanus; another causes tremors, convulsions, and subsequently paralysis; and a third causes at once intense clonic and tonic spasms. Another point of interest is the fact that protection in animals can be procured by inoculating an animal with the blood of another which has had the disease. The organism has been found in the earth and in putrefying fluids, and Nicolaïer has caused the disease by inoculating with different sorts of surface soil.

Morbid Anatomy.—No characteristic lesions have been found in the cord or in the brain. Congestions occur in different parts, and perivascular exudations and granular changes in the nerve-cells have been found. The condition of the wound is variable. The nerves are often found injured, reddened, and swollen. In the tetanus neonatorum the umbilicus may be inflamed.

Symptoms.—After an injury the disease sets in usually within ten days. In Yandell's statistics at least two fifths, and in Joseph Jones's four fifths, occurred before the fifteenth day. The patient complains at first of slight stiffness in the neck, or a feeling of tightness in the jaws, or difficulty in mastication. Occasionally chilly feelings or actual rigors may precede these symptoms. Gradually a tonic spasm of the muscles of these parts develops, producing the condition of trismus or lockjaw. The eyebrows may be raised and the angles of the mouth drawn out, causing the so-called sardonic grin—*risus sardonicus*. In children the spasm may be confined to these parts. Sometimes the attack is associated with paralysis of the facial muscles and difficulty in swallowing—the head tetanus of Rose, which has most commonly followed injuries in the neighborhood of the fifth nerve. Gradually the paroxysms extend and involve the muscles of the body. Those of the back are most affected, so that during the spasm the unfortunate victim may rest upon the head and heels, a position known as *opisthotonos*. The rectus abdominalis muscle has been torn across in the spasm. The entire trunk and limbs may be perfectly rigid—*orthoto-*

nos. Flexion to one side is less common—*pleurosthotonos;* while spasm of the muscles of the abdomen may cause the body to be bent forward—*emprosthotonos.* In very violent attacks the thorax is compressed, the respirations are rapid, and spasm of the glottis may occur, causing asphyxia. The paroxysms last for a variable period, but even in the intervals the relaxation is not complete. The slightest irritation is sufficient to cause a spasm. The paroxysms are associated with agonizing pain, and the patient may be held as in a vise, unable to utter a word. Usually he is bathed in a profuse sweat. The temperature may remain normal throughout, or show only a slight elevation toward the close. In other cases the pyrexia is marked from the outset; the temperature reaches 105° or 106°, and before death 109° or 110°. In rare instances the temperature may reach a still higher point. Death either occurs during the paroxysm from heart-failure or asphyxia, or is due to exhaustion.

Diagnosis.—Well-developed cases following a trauma could not be mistaken for any other disease. The spasms are not unlike those of strychnia-poisoning, and in the celebrated Palmer murder trial this was the plea for the defence. The jaw-muscles, however, are never involved early, if at all, and between the paroxysms in strychnia-poisoning there is no rigidity. Certain cases of the so-called cephalic tetanus in which there is difficulty of swallowing might be mistaken for hydrophobia, but in this disease there is never the stiffness of the jaws nor paroxysms in which the cervical and dorsal muscles are affected. In tetany the distribution of the spasm at the extremities, the peculiar position, the greater involvement of the hands, and the condition under which it occurs, are sufficient to make the diagnosis clear.

Prognosis.—Two of the Hippocratic aphorisms express tersely the general prognosis even at the present day: "The spasm supervening on a wound is fatal," and "such persons as are seized with tetanus die within four days, or if they pass these they recover."

The mortality in the traumatic cases is not less than eighty per cent. (Conner); in the idiopathic cases it is under fifty per cent. According to Yandell the mortality is greatest in children. Favorable indications are —late onset of the attack, localization of the spasms to the muscles of the neck and jaw, and an absence of fever. Most of the cases of Rose's head tetanus, the so-called *tetanus hydrophobicus*, recover.

Treatment.—The patient should be kept in a darkened room, absolutely quiet, and attended by only one person. All possible sources of irritation should be avoided. Veterinarians appreciate the importance of this complete seclusion, and in well-equipped infirmaries there may be seen a brick padded chamber in which these cases are treated.

When the lockjaw is extreme it may be impossible to feed the patient, under which circumstances it is best to use rectal injections, or to feed by a catheter passed through the nose. The spasm should be controlled by chloroform, which may be repeatedly given at intervals. It is more satis-

factory to keep the patient thoroughly under the influence of morphia given hypodermically. Chloral hydrate, bromide of potassium, Calabar bean, curara, Indian hemp, belladonna, and other drugs have been recommended, and recovery occasionally follows their use. As the toxic agents appear to be produced by bacilli at the site of the lesion, thorough cleansing and antiseptic treatment should be carried out.

XXV. SYPHILIS.

Definition.—A specific disease of slow evolution, propagated by inoculation (acquired syphilis), or by hereditary transmission (congenital syphilis). In the acquired form the site of inoculation becomes the seat of a special tissue change—*primary lesion.* After an interval of two or three months constitutional symptoms develop, with affections of the skin and mucous membranes—*secondary lesions.* And, finally, after a period of three, four, or more years, granulomatous growths develop in the viscera, muscles, bones, or skin—*tertiary lesions.*

I. General Etiology and Morbid Anatomy.

The nature of the virus is still doubtful. Lustgarten found in the hard chancre and in gummata a rod-shaped bacillus of 3 or 4 μ in length, which he claims is specific and peculiar to the disease. This organism closely resembles the smegma bacillus, which is found beneath the prepuce, but from its occurrence in gummatous growths it is hardly possible that they can be identical. Further observations are required before the question can be considered settled.

Syphilis is peculiar to man, and cannot be transmitted to the lower animals. All are susceptible to the contagion, and it occurs at all ages.

Modes of Infection.—(1) In a large majority of all cases the disease is transmitted by *sexual congress*, but the designation *venereal* disease, *lues venerea*, is not always correct, as there are many other modes of inoculation.

(2) *Accidental Infection.*—In surgical and in midwifery practice, physicians are not infrequently inoculated. It is surprising that infection from these sources is not more common. I have known personally of six cases. Midwifery chancres are usually on the fingers, but I have met with one instance on the back of the hand. Lip, mouth, and tonsillar sores result as a rule from improper practices. Wet-nurses are sometimes infected on the nipple, and it occasionally happens that relatives of the child are accidentally contaminated. One of the most lamentable forms of accidental infection is the transmission of the disease in humanized vaccine lymph. This, however, is extremely rare. The conditions under which it occurs have been already referred to (see Vaccination).

(3) *Hereditary Transmission.*—This may be, and is most common,

from (*a*) the father, the mother being healthy (sperm inheritance). It is, unfortunately, an every-day experience to see cases of congenital syphilis in which the infection is clearly paternal. A syphilitic father may, however, beget a healthy child, even when the disease is fresh and full-blown. On the other hand, in very rare instances, a man may have had syphilis when young, undergo treatment, and for years present no signs of disease, and yet his first-born may show very characteristic lesions. Happily, in a large majority of instances, when the treatment has been thorough, the offspring escape. The closer the begetting to the primary sore, the greater the chance of infection. A man with tertiary lesions may beget healthy children. As a general rule it may be said that with judicious treatment the transmissive power rarely exceeds three or four years.

(*b*) Maternal transmission (germ inheritance). It is a remarkable and interesting fact that a woman who has borne a syphilitic child is herself immune, and cannot be infected, though she may present no signs of the disease. This is known as Colles's law, and was thus stated by the distinguished Dublin surgeon: "That a child born of a mother who is without obvious venereal symptoms, and which, without being exposed to any infection subsequent to its birth, shows this disease when a few weeks old—this child will infect the most healthy nurse, whether she suckle it or merely handle and dress it; and yet this child is never known to infect its own mother, even though she suckle it while it has venereal ulcers of the lips and tongue." In a majority of these cases the mother has received a sort of protective inoculation, without having had actual manifestations of the disease.

A woman with acquired syphilis is liable to bear infected children. The father may not be affected. In a large number of instances both parents are diseased, the one having infected the other, in which case the chances of fœtal infection are greatly increased.

(*c*) Placental transmission. The mother may be infected after conception, in which case the child may be, but is not necessarily, born syphilitic.

Morbid Anatomy.—The *primary lesion*, or chancre, shows: (*a*) A diffuse infiltration of the connective tissue with small, round cells. (*b*) Larger epithelioid cells. (*c*) Giant cells. (*d*) The Lustgarten bacilli, in small numbers. (*e*) Changes in the small arteries, chiefly thickening of the intima, and alterations in the nerve-fibres going to the part (Berkeley). The sclerosis is due in part to this acute obliterative endarteritis. Associated with the initial lesions are changes in the adjacent lymph-glands, which undergo hyperplasia, and finally become indurated.

The *secondary lesions* of syphilis are too varied for description here. They consist of condylomata, skin eruptions, affections of the eye, etc.

The *tertiary lesions* consist of circumscribed tumors known as gummata, and of an arteritis, which, however, is not peculiar to the disease.

Gummata.—Syphilomata develop in the bones or periosteum—here

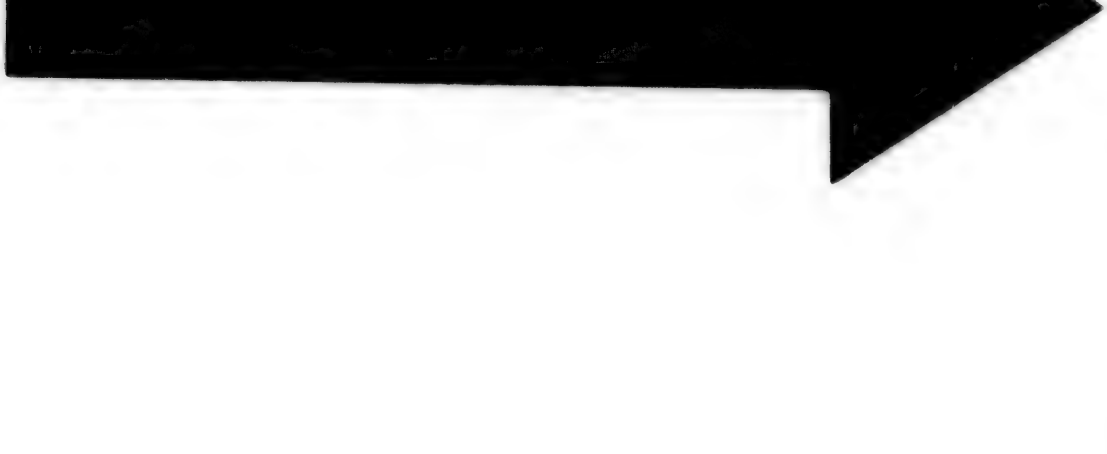
they are called nodes—in the muscles, skin, brain, lung, liver, kidneys, heart, testes, and adrenals. They vary in size from small, almost microscopic, bodies to large, solid tumors from three to five centimetres in diameter. They are usually firm and hard, but in the skin and on the mucous membranes they tend to break down rapidly and ulcerate. On cross-section a medium-sized gumma has a grayish-white, homogeneous appearance, presenting in the centre a firm, caseous substance, and at the periphery a translucent, fibrous tissue. Often there are groups of three or more surrounded by dense sclerotic tissue. They are usually very firm and hard. Histologically, a small gumma consists of a granulation tissue composed of rounded cells. Owing to insufficient blood-supply, coagulation necrosis takes place in the centre with the formation of a fibro-caseous material, while the growth extends at the margins with the gradual production of fibre-cells. Ultimately the central caseous part may be absorbed, and healing takes place with the development of a fibrous scar.

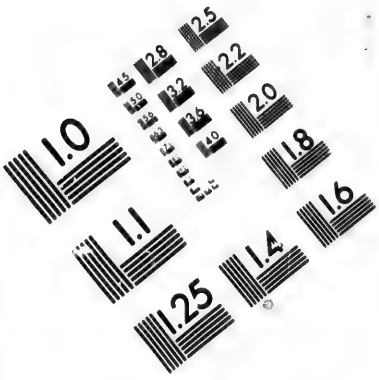
The arteritis will be considered in a separate section.

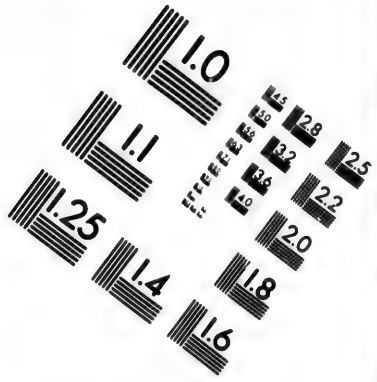
II. Acquired Syphilis.

Primary Stage.—This extends from the appearance of the initial sore until the onset of the constitutional symptoms, and has a variable duration of from six to twelve weeks. The initial sore appears within a month after inoculation, and it first shows itself as a small red papule, which gradually enlarges and breaks in the centre, leaving a small ulcer. The tissue about this becomes indurated so that it ultimately has a gristly, cartilaginous consistence—hence the name, hard or indurated chancre. The size attained is variable, and when small the sore may be overlooked, particularly if it is just within the urethra. The glands in the lymph-district of the chancre enlarge and become hard. Suppuration both in the initial lesion and in the glands may occur as a secondary change. The general condition of the patient in this stage is good. There may be no fever and no impairment of health.

Secondary Stage.—The first constitutional symptoms are usually manifested within three months of the appearance of the primary sore. They rarely develop earlier than the sixth or later than the twelfth week. The symptoms are: (*a*) *Fever*, slight or intense, and very variable in character. A mild continuous pyrexia is not uncommon, the temperature not rising above 101°. The fever may have a distinctly remittent character; but the most remarkable and puzzling type of syphilitic fever is the intermittent, which often leads to error in diagnosis. The fever may come on within a month after exposure and rise to 104° or 105°, with oscillations of five or six degrees (Yeo) A remarkable case is reported by Sidney Phillips, in which pyrexia persisted for months, with paroxysms resembling in all respects tertian ague, and which resisted quinine and yielded promptly to mercury and potassium iodide. Although usually a secondary manifestation, the fever of syphilis may occur late in the disease.







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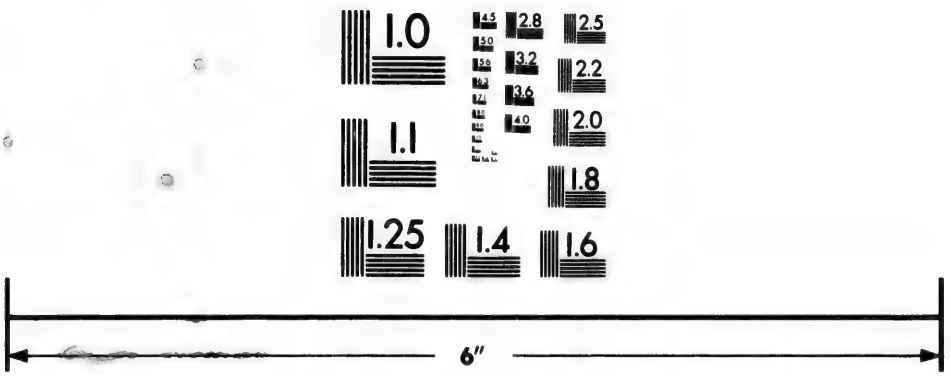

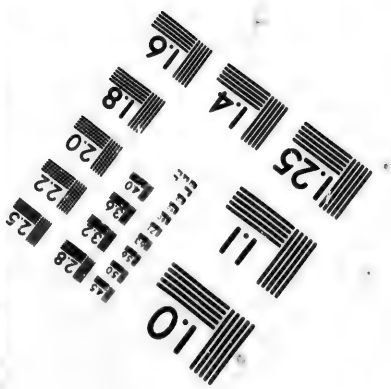

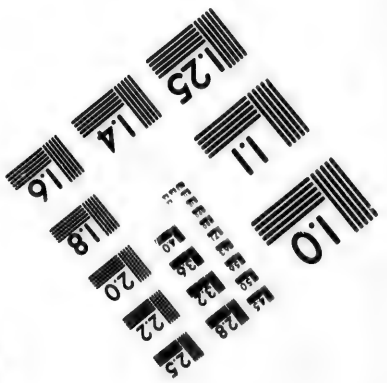

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(*b*) *Anæmia.*—In many cases the syphilitic poison causes a pronounced anæmia which gives to the face a muddy pallor, and there may even be a light-yellow tingeing of the conjunctivæ or of the skin, an hæmatogenous icterus. This syphilitic cachexia may in some instances be extreme. The red blood-corpuscles do not show any special alterations. The blood-count may fall to three millions per cubic millimetre, or even lower, and the hæmoglobin to forty or fifty per cent (Hayem). No characteristic organisms have been found in the blood.

(*c*) *Cutaneous Lesions.*—Skin eruptions of all forms may develop. The earliest and most common is a rash—*macular syphilide* or *syphilitic roseola*—which occurs on the abdomen, the chest, and on the front of the arms. The face is often exempt. The spots, which are reddish-brown and symmetrically arranged, persist for a week or two. Next in frequency is a *papular syphilide*, which may form acne-like indurations about the face and trunk, often arranged in groups. Other forms are the *pustular rash*, which may so closely simulate variola that the patient may be sent to a small-pox hospital. A *squamous syphilide* occurs, not unlike ordinary psoriasis, except that the scales are less abundant. The rash is more copper-colored and not specially confined to the extensor surfaces.

In the moist regions of the skin, such as the perinæum and groins, the axillæ, between the toes, and at the angles of the mouth, the so-called *mucous patches* develop, which are flat, warty outgrowths, with well-defined margins and surfaces covered with a grayish secretion. They are among the most distinctive lesions of syphilis.

Frequently the hair falls out (alopecia), either in patches or by a general thinning. Occasionally the nails become affected (syphilitic onychia).

(*d*) *Mucous Lesions.*—With the fever and the roseolous rash the throat and mouth become sore. The pharyngeal mucosa is hyperæmic, the tonsils are swollen and often present small, kidney-shaped ulcers with grayish-white borders. Mucous patches are seen on the inner surfaces of the cheeks and on the tongue and lips. Sometimes on the tongue there are whitish spots (leucomata), which are seen most frequently in smokers, and which Hutchinson regards as the joint result of syphilitic glossitis and the irritation of hot tobacco-smoke. Hypertrophy of the papillæ in various portions of the mucous membrane produces the syphilitic warts or condylomata which are most frequent about the vulva and anus.

(*e*) *Other Lesions.*—*Iritis* is common, and usually affects one eye before the other. It develops in from three to six months after the chancre. There may be only slight ciliary congestion in mild cases, but in severer forms there is great pain, and the condition is serious and demands careful management. *Choroiditis* and *retinitis* are rare secondary symptoms. Ear affections are not common in the secondary stage, but instances are found in which sudden deafness develops, which may be due to labyrinthine disease; more commonly the impaired hearing is due to the extension

of inflammation from the throat to the middle ear. Epididymitis is an occasional secondary lesion.

Tertiary Stage.—No hard and fast line can be drawn between the lesions of the secondary and those of the tertiary period; and, indeed, in exceptional cases, manifestations which usually appear late may set in even before the primary sore has properly healed. The special affections of this stage are certain skin eruptions, gummatous growths in the viscera, and amyloid degenerations.

(*a*) The late *syphilides* show a greater tendency to ulceration and destruction of the deeper layers of the skin, so that in healing scars are left. They are also more scattered and seldom symmetrical. One of the most characteristic of the tertiary syphilides is rupia, the dry stratified crusts of which cover an ulcer which involves the deeper layers of the skin and in healing leaves a scar.

(*b*) *Gummata.*—These may develop in the skin, subcutaneous tissue, muscles, or internal organs. The general character has been already described. When they develop in the skin they tend to break down and ulcerate, leaving ugly sores which heal with difficulty. In the solid organs they undergo fibroid transformation and produce puckering and deformity. On the mucous membranes these tertiary lesions lead to ulceration, in the healing of which cicatrices are formed; thus, in the larynx great narrowing may result, and in the rectum ulceration with fibroid thickening and retraction may lead to stricture.

(*c*) *Amyloid Degeneration.*—Syphilis plays a most important *rôle* in the production of this affection. Of 244 instances analyzed by Fagge, 76 had syphilis, and of these 42 had no bone lesions. It follows the acquired form and is very common in the rectal disease in women. In congenital lues amyloid degeneration is rare.

(*d*) *Sclerosis*—Syphilis is an important factor in inducing degenerative changes in certain tissues. In locomotor ataxia the association between this disease and sclerosis of the posterior columns of the cord is far too frequent to be accidental, but the precise relations cannot, with our present knowledge, be explained. With regard to arterio-sclerosis, the part played by syphilis is unquestioned, but the nature of the connection of the two processes remains doubtful.

III. Congenital Syphilis.

With the exception of the primary sore, every feature of the acquired disease may be seen in the congenital form.

The intra-uterine conditions leading to the death of the fœtus do not here concern us. The child may be born healthy-looking, or with well-marked evidences of the disease. In the majority of instances the former is the case, and within the first month or two the signs of the disease appear.

Symptoms.—(*a*) *At Birth.*—When the disease exists at birth the

child is feebly developed and wasted, and a skin eruption is usually present, commonly in the form of bullæ about the wrists and ankles, and on the hands and feet (pemphigus neonatorum). The child snuffles, the lips are ulcerated, the angles of the mouth fissured, and there is enlargement of the liver and spleen. The bone symptoms may be marked, and the epiphyses may even be separated. In such cases the children rarely survive long.

(*b*) *Early Manifestations.*—When born healthy the child thrives, is fat and plump, and shows no abnormity whatever; then from the fourth to the eighth week, rarely later, a nasal catarrh develops, *syphilitic rhinitis*, which impedes respiration, and produces the characteristic symptom which has given the name *snuffles* to the disease. The discharge may be sero-purulent or bloody. The child nurses with great difficulty. In severe cases ulceration takes place with necrosis of the bone, leading to a depression at the root of the nose and a deformity characteristic of congenital syphilis. This coryza may be mistaken at first for an ordinary catarrh, but the coexistence of other manifestations usually makes the diagnosis clear. The disease may extend into the Eustachian tubes and middle ear and lead to deafness.

The *cutaneous lesions* develop with or shortly after the onset of the snuffles. The skin often has a sallow, earthy hue. The eruptions are first noticed about the nates. There may be an erythema or an eczematous condition, but more commonly there are irregular reddish-brown patches with well-defined edges. A papular syphilide in this region is by no means uncommon. Fissures develop about the lips, either at the angles of the mouth or in the median line. These *rhagades*, as they are called, are very characteristic. There may be marked ulceration of the mucocutaneous surfaces. The secretions from these mouth lesions are very virulent, and it is from this source that the wet-nurse is usually infected. Not only the nurse, but members of the family, may be contaminated. There are instances in which other children have been accidentally inoculated from a syphilitic infant. The hair of the head or of the eyebrows may fall out. The syphilitic *onychia* is not uncommon. Enlargement of the glands is not so frequent in the congenital as in the acquired disease. When the cutaneous lesions are marked, the contiguous glands can usually be felt. As pointed out by Gee, the spleen is enlarged in many cases. The condition may persist for a long time. Enlargement of the liver, though often present, is less significant, since in infants it may be due to various causes. These are among the most constant symptoms of congenital syphilis, and usually develop between the third and twelfth weeks. Frequently they are preceded by a period of restlessness and wakefulness, particularly at night. Some authors have described a peculiar syphilitic cry, high-pitched and harsh. Among rarer manifestations are hæmorrhages—the *syphilis hæmorrhagica neonatorum*. The bleeding may be subcutaneous, from the mucous surfaces, or, when early, from the

umbilicus. All of such cases, however, are not syphilitic, and the disease must not be confounded with the acute hæmoglobinuria of new-born infants, which Winckel describes as occurring in epidemic form, and which is probably an acute infectious disorder.

(*c*) *Late Manifestations.*—Children with congenital syphilis rarely thrive. Usually they present a wizened, wasted appearance, and a prematurely aged face. In the cases which recover, the general nutrition may remain good and the child may show no further manifestations of the disease; commonly, however, at the period of second dentition or at puberty the disease reappears. Although the child may have recovered from the early lesions, it does not develop like other children. Growth is slow, development tardy, and there are facial and cranial characteristics which often render the disease recognizable at a glance. A young man of nineteen or twenty may neither look older nor be more developed than a boy of ten or twelve. Fournier describes this condition as *infantilism.* The forehead is prominent, the frontal eminences are marked, and the skull may be very asymmetrical. The bridge of the nose is depressed, the tip *retroussé.* The lips are often prominent, and there are striated lines running from the corners of the mouth The *teeth* are deformed and may present appearances which Jonathan Hutchinson claims are specific and peculiar. The upper central incisors of the permanent set are the teeth which give information. The specific alterations are—the teeth are peg-shaped, stunted in length and breadth, and narrower at the cutting edge than at the root. On the anterior surface the enamel is well formed, and not eroded or honeycombed. At the cutting edge there is a single notch, usually shallow, sometimes deep, in which the dentine is exposed.

Among late manifestations, particularly apt to appear about puberty, is the interstitial *keratitis*, which usually begins as a slight steaminess of the corneæ, which present a ground-glass appearance. It affects both eyes, though one is attacked before the other. It may persist for months, and usually clears completely, though it may leave opacities, which prevent clear vision. *Iritis* may also occur. Of *ear affections*, apart from those which develop as a sequence of the pharyngeal disease, a form occurs about the time of puberty or earlier, in which deafness comes on rapidly and persists in spite of all treatment. It is unassociated with obvious lesions, and is probably labyrinthine in character. *Bone lesions*, occurring oftenest after the sixth year, are not rare among the late manifestations of hereditary syphilis. The tibiæ are most frequently attacked. It is really a chronic gummatous periostitis, which gradually leads to great thickening of the bone. The nodes of congenital syphilis, which are often mistaken for rickets, are more commonly diffuse and affect the bones of the upper and lower extremities. They are generally symmetrical and rarely painful. They may develop late, even after the twenty-first year.

Joint lesions are rare. Clutton has described a symmetrical synovitis

of the knee in hereditary syphilis. Lastly, it must be borne in mind that enlargement of the spleen may be one of the late manifestations, and may occur either alone or in connection with disease of the liver. At the University Hospital, Philadelphia, I had under observation for more than a year a girl of thirteen, small and feebly developed, with a luetic facies, whose spleen reached as low as the level of the navel. The condition was not thought to be due to inherited syphilis until she developed osseous lesions.

Gummata of the liver, brain, and kidneys have been found in late hereditary syphilis.

IV. Visceral Syphilis.

A. **Syphilis of the Brain and Cord.**—The following lesions occur:

(1) *Gummata*, forming definite tumors, ranging in size from a pea to a walnut. They are usually multiple and attached to the pia mater, sometimes to the dura. Very rarely they are found unassociated with the meninges. When small they present a uniform, translucent appearance, but when large the centre undergoes a fibro-caseous change, while at the periphery there is a firm, translucent, grayish tissue. They may closely resemble large tuberculous tumors. The growths are most common in the cerebrum. They may be multiple and may even attain a considerable size without becoming caseous. Occasionally gummata undergo cystic degeneration. In the cord large gummatous growths are not so common. In an instance recently reported by me a tumor, from three eighths to one fourth of an inch in diameter, was completely within the cord opposite the fourth cervical nerve, and there were numerous gummata in the cauda equina.

(2) *Gummatous Meningitis.*—This constantly occurs in the neighborhood of the larger growths, and there may be local meningeal thickening several centimetres in extent, in which the pia is infiltrated and the arteries greatly thickened. This by no means uncommon form may run a subacute or a chronic course.

(3) *Gummatous Arteritis.*—The lesions may be confined to the arteries which present the nodular tumors to be described hereafter.

(4) Foci of *sclerosis*, which Lancereaux holds may be distinguished from non-specific forms by a much greater tendency of the neuroglia elements to undergo fatty transformation, and by the secondary alterations, as areas of softening, which occur in the neighborhood. Neither the diffuse nor the nodular cerebral sclerosis, met with particularly in children, appears to have any special relation to inherited syphilis.

(5) Whether a localized encephalitis or myelitis can result from the action of the syphilitic poison without involvement of the blood-vessels is doubtful. In a case of multiple arterial gummata recently in my ward, Thomas found in the lumbar region of the cord foci of inflammatory softening.

Secondary Changes.—In the brain gummatous arteritis is one of the common causes of softening, which may be extensive, as when the middle cerebral artery is involved, or when there is a large patch of syphilitic meningitis. In such instances the process is really a meningo-encephalitis, and the symptoms are due to the secondary changes in the brain-substance, not directly to the gumma. In the neighborhood of a gummatous growth intense encephalitis or myelitis may develop, and within a few days change the clinical picture. Gummatous arteritis may lead to weakening of the wall of the vessel and rupture with meningeal hæmorrhage.

Syphilitic disease of the nerve-centres may occur in the inherited or acquired form, most commonly in the latter. In the congenital cases the tumors usually develop early, but may be as late as the twenty-first year (H. C. Wood). In the acquired form the nerve lesions belong, as a rule, to the late manifestations, and patients may have quite forgotten the existence of a primary infection, and in very many instances the secondary manifestations have been slight. Heubner, to whom we owe so much in connection with this subject, has seen it as late as the thirtieth year. On the other hand, in exceptional instances, it may occur very early, and convulsions and brain symptoms have been reported within three months of the primary sore. In one of my cases, in which there was endarteritis followed by dilatation and perforation, the patient had had a hard chancre eighteen months before, with severe secondary symptoms.

Symptoms.—The chief features of cerebral syphilis are those of tumor, which will be considered subsequently under that section. They may be classified here as follows:

(1) Psychical features. A sudden and violent onset of delirium may be the first symptom. In other instances prior to the occurrence of delirium there have been headache, alteration of character, and loss of memory. The condition may be accompanied by convulsions. There may be no neuritis, no palsy, and no localizing symptoms.

(2) More commonly following headache, giddiness, or an excited state which may amount to delirium, the patient has an epileptic seizure or develops hemiplegia, or there is involvement of the nerves of the base. Some of these cases display a prolonged torpor, a special feature of brain syphilis to which both Buzzard and Heubner have referred, which may persist for as long as a month. H. C. Wood describes with this a state of automatism occurring particularly at night, in which the patient behaves like "a restless nocturnal automaton rather than a man."

(3) A clinical picture of general paralysis—dementia paralytica. The question is still in dispute whether this syphilitic encephalopathy, which so closely resembles general paralysis, is a distinct and independent affection. Mickle, who has carefully reviewed the subject, concludes that syphilis may directly produce the inflammatory changes in the brain, while in other instances it directly predisposes to this affection. It is a somewhat remarkable feature that the cases which present the clinical picture

of general paresis are most frequently those which have not had any focalizing symptoms, and they may not have convulsions until the disease is well advanced. Another peculiarity is the fact that, like the late sclerosis, the condition is not very amenable to the specific treatment; though Mitchell mentions an instance which he regarded as general paresis, but which subsequently came into the hands of H. C. Wood, who cured the case with iodide of potassium.

(4) Many cases of cerebral syphilis display the symptoms of brain tumor—headache, optic neuritis, vomiting, and convulsions. Of these symptoms convulsions are the most important, and both Fournier and Wood have laid great stress on the value of this symptom in persons over thirty. The first symptoms may, however, rather resemble embolism or thrombosis; thus there may be sudden hemiplegia, with or without loss of consciousness.

The symptoms of *spinal syphilis* are extremely varied and may be caused either by large gummatous growths attached to the meninges, in which case the features are those of tumor; or by meningitis with secondary spastic changes; or again by scleroses developing late in the disease, the relation of which to syphilis is still obscure.

Diagnosis.—The history is of the first importance, but it may be extremely difficult to get a reliable account. Careful examination should be made for traces of the primary sore, for the cicatrices of bubo, for scars of the skin eruption or throat ulcers, and for bone lesions. The character of the symptoms is often of great assistance. They are multiform, variable, and often such as could not be explained by a single lesion; thus there may be anomalous spinal symptoms or involvement of the nerves of the brain on both sides. And lastly the result of treatment has a definite bearing on the diagnosis, as the symptoms may clear up and disappear with the use of antisyphilitic remedies.

B. Syphilis of the Lung.

This is a very rare disease. During twenty years I have not seen more than half a dozen specimens in which there was no question as to the nature of the trouble. Early in my professional life I learned to recognize the disease from the teaching of Wilks, and became familiar with the excellent specimens preserved at Guy's Hospital. In my ten years' work in Montreal not a single specimen was recognized at the dissections at the General Hospital. In 1878 and 1884 I saw several characteristic examples in London and Germany. During five years in Philadelphia, for the greater part of which time I was connected with the Philadelphia Hospital, which has perhaps as rich luetic material as is to be found anywhere, only one or two specimens were seen. Three admirable illustrations of pulmonary gummata have occurred at the Johns Hopkins Hospital during the past two years. I mention these details because the subject is one which has always interested me, and I have been constantly on the lookout for the disease. It has been a continual surprise that it should be so com-

mon in certain localities, but I find that my experience as to its comparative rarity tallies very closely with that of pathologists and hospital physicians in this country and in Europe. The literature of the subject is extensive, but from the clinical aspect largely worthless, as it preceded Koch's discovery of the *bacillus tuberculosis*.

Etiology and Morbid Anatomy.—Syphilis of the lung occurs under the following forms:

(1) The *white pneumonia of the fœtus*. This may affect large areas or an entire lung, which then is firm, heavy and airless, even though the child may have been born alive. On section it has a grayish-white appearance—the so-called white hepatization of Virchow. The chief change is in the alveolar walls, which are greatly thickened and infiltrated, so that, as Wagner expressed it, the condition resembles a diffuse syphiloma. In the early stages, for example, in a seven or eight months' fœtus, there may be scattered miliary foci of this induration chiefly about the arteries. The air-cells are filled with desquamated and swollen epithelium.

(2) In the form of definite *gummata*, which vary in size from a pea to a goose-egg. They occur irregularly scattered through the lung, but, as a rule, are more numerous toward the root. They present a grayish-yellow caseous appearance, are dry and usually imbedded in a translucent, more or less firm, connective tissue. In a case from my wards recently described by Councilman, there was extensive involvement of the root of the lungs. Bands of connective tissue passed inward from the thickened pleura and between these strands and surrounding the gummata there was in places a mottled red pneumonic consolidation. In the caseous nodules there is typical hyaline degeneration. Councilman describes as the primary lesion, atrophy of the alveolar walls with hyaline degeneration of the capillaries, not the syphilitic endarteritis, which is well marked, and to which the lesions are attributed. The bronchi are usually involved, and surrounding the gummata there may be a diffuse broncho-pneumonia, which does not appear to have any peculiar characters.

(3) A majority of authors follow Virchow in recognizing the fibrous interstitial pneumonia at the root of the lung and passing along the bronchi and vessels as probably syphilitic. This much may be said, that in certain cases gummata are associated with these fibroid changes. Again, this condition alone is found in persons with well-marked syphilitic history or with other visceral lesions. It seems in many instances to be a purely sclerotic process, advancing sometimes from the pleura, more commonly from the root of the lung, and invading the interlobular tissue, gradually producing a more or less extensive fibroid change. It rarely involves more than a portion of a lobe or portions of the lobes at the root of the lung. The bronchi are often dilated.

Symptoms.—Is there a syphilitic phthisis, an ulcerative and destructive disease, due to lues? Personally I have no knowledge of such an affection, either clinically or anatomically, and the cases which I have seen

demonstrated do not seem to me to have characters distinctive enough to separate them from ordinary tuberculous phthisis. Certain French writers recognize not only a chronic syphilitic phthisis but an acute syphilitic pneumonia in adults, simulating acute pneumonic phthisis. Clinically, pulmonary syphilis is not of much importance, as the cases can rarely be diagnosed, and the symptoms which arise are usually those of bronchiectasis or of chronic interstitial pneumonia. The white pneumonia is usually found in the still-born.

Diagnosis.—It is to be borne in mind, in the first place, that hospital physicians and pathologists the world over bear witness to the extreme rarity of lung syphilis. In the second place, the therapeutic test upon which so much reliance is placed is by no means conclusive. With pulmonary tuberculosis there should now be no confusion, owing to the readiness with which the presence of bacilli is determined. Bronchiectasy in the lower lobe of a lung, dependent upon an interstitial pneumonia of syphilitic origin, could not be distinguished from any other form of the disease. In persons with well-marked syphilitic lesions elsewhere, when obscure pulmonary symptoms occur, or if there are signs of chronic interstitial pneumonia with dilated bronchi, and no tubercle bacilli are present, the condition may possibly be due to syphilis. So far as my experience goes, tuberculous phthisis occurring in a syphilitic subject has no special peculiarities. The lesions of syphilis and tuberculosis could of course coexist in a lung. Since writing the above, the recent paper of Satterthwaite has appeared, ·t one of the cases upon which it is based could properly be regard· yphilitic in the absence of an examination for tubercle bacilli. Much more suggestive of true syphilitic phthisis is Case I of McLane Tiffany's series, but it too may have been tuberculous. It is quite possible that a large caseous gumma may break down and form a cavity, but the existence of an extensive ulcerative and destructive disease of the lungs (comparable to tuberculosis) due to syphilis has not yet been proved.

c. **Syphilis of the Liver.**

This occurs in three forms: (*a*) *Diffuse Syphilitic Hepatitis.*—This is most common in cases of congenital syphilis. The liver preserves its form, is large, hard, and resistant. Sometimes it has a yellow look, compared by Trousseau to sole-leather, or an appearance not unlike the amyloid liver. Careful inspection shows grayish or whitish points and lines corresponding to the interlobular new growth. Microscopically, great increase in the connective tissue is seen, and in many places foci of small-celled infiltration. Sometimes these nodules are visible, forming firm miliary gummata which in cicatrizing produce more or less deformity. Larger gummata may also be present.

(*b*) *Gummata.*—As a result of congenital syphilis these may occur in childhood or in adult life. In acquired syphilis they rarely come on before the second year after infection. In the early stage there are pale

grayish nodules, varying in size from a pea to a marble. The larger, which are usually limited toward the liver tissue, present yellowish centres at first; but later there is a "pale yellowish, cheese-like nodule of irregular outline, surrounded by a fibrous zone, the outer edge of which loses itself in the lobular tissue, the lobules dwindling gradually in its grasp. This fibrous zone is never very broad; the cheesy centre varies in consistence from a gristle-like toughness to a pulpy softness; it is sometimes mortar-like, from cretaceous change" (Wilks). When numerous, the most extensive deformity of the liver is produced in the gradual healing of these gummata. On the surface there are deep, scar-like depressions, and the entire organ may be divided into a cluster of irregular masses, held together by fibrous tissue. To this condition the term *botyroid* has been given, from its resemblance to a bunch of grapes. As a rule, the gummata gradually undergo fibroid transformation. They may, however, soften and liquefy, and, according to Wilks, may form a fluctuating tumor.

(*c*) Occasionally the syphilitic changes are chiefly manifested in *Glisson's sheath*, in a thickening of the capsule, producing peri-hepatitis, and increase in the connective tissue in the *portal canals*, so that on section the organ presents a number of branching fibrous scars which may cause considerable deformity.

Symptoms.—The symptoms of syphilitic hepatitis are very variable. In the new-born icterus is not uncommon, but the condition of the liver can scarcely be recognized. In the adult there are two groups of cases:

The patient presents a picture of cirrhosis of the liver; there are digestive disturbances, slight icterus, loss of weight, and ascites. If signs of syphilis are present in other organs, the condition may be suspected, or if after removal of the fluid the liver is felt to be extremely irregular, the diagnosis may be made almost with certainty. As these cases, with proper treatment, may recover, they form a certain contingent of the cases reported as recovery in ordinary cirrhosis of the liver.

In a second group of cases the patient is anæmic, passes large quantities of pale urine containing albumen and tube-casts; the liver is enlarged, perhaps irregular, and the spleen also is enlarged. Dropsical symptoms may supervene, or the patient may be carried off by some intercurrent disease. Extensive amyloid degeneration of the spleen, the intestinal mucosa, and of the liver, with gummata, are found.

The *diagnosis* of syphilis of the liver is very important, since upon it the proper treatment depends. If with a history of infection the liver is enlarged and irregular, and the general health fairly good, the condition is probably syphiloma. Occasionally tumors of a definite form may be produced by the gummata. For two years I showed repeatedly, at my clinic at the University Hospital, Philadelphia, a boy aged eleven, who had a prominent tumor in the epigastrium connected with the liver, the nature of which was obscure until well-marked bone-lesions developed.

In another case, a man, aged thirty, was sent to me for advice concerning the making of an exploratory incision to determine the nature of a firm, irregular tumor which occupied the epigastric region, and was evidently connected with the left lobe of the liver. It had lasted for more than a year, had increased slightly, and had not impaired, to any marked degree, the general health. This fact, together with a well-marked history of acquired syphilis, led me to place him upon a rigid antisyphilitic treatment, with the result that within six months the entire tumor disappeared.

D. **Syphilis of the Digestive Tract.**

The *œsophagus* is very rarely affected. Stenosis is the usual result. Gummata of the *stomach* occur occasionally. Syphilitic ulceration has been found in the stomach, in the small intestine, and in the cæcum.

The most common seat of syphilitic disease in this tract is the *rectum*. The affection is found most commonly in women, and results from the development of gummata in the submucosa above the internal sphincter. The process is slow and tedious, and may last for years before it finally induces stricture. The symptoms are usually those of narrowing of the lower bowel. The condition is readily recognized by rectal examination. The history of gradual on-coming stricture, the state of the patient, and the fact that there is a hard, fibrous narrowing, not an elevated crater-like ulcer, usually render easy the diagnosis from malignant disease. In medical practice these cases come under observation for other symptoms, particularly amyloid degeneration; and the rectal disease may be entirely overlooked, and only discovered post mortem.

E. **Circulatory System.**

Syphilis of the Heart.—A fresh, warty endocarditis due to syphilis is not recognized, though occasionally in persons dead of the disease this form is present, as is not uncommon in conditions of debility. Outgrowths on the valves in connection with gummata have been reported by Janeway and others, and in Lang's* monograph there are thirteen cases which he reports as syphilitic endocarditis, most of them of the fibrous or sclerotic variety.

Syphilitic myocarditis appears either in the form of diffuse fibroid induration or as definite gummata. Lang has collected many cases from the literature, a majority of which were of the former description. Gummata, however, occur not infrequently as definite and characteristic tumors in the myocardium. Rupture may take place, as in the cases reported by Dandridge and Nalty, or sudden death, as in the cases of Cayley and Pearce Gould.

Syphilis of the Arteries.—Syphilis is believed to play an important *rôle* in arterio-sclerosis and aneurism. Its connection with these processes will be considered later; here we shall refer only to the syphilitic arteritis. This occurs in two forms:

* Die Syphilis des Herzens, Wien, 1889.

(*a*) An *obliterating endarteritis*, characterized by a proliferation of the subendothelial tissue. The new growth lies within the elastic lamina, and may gradually fill the entire lumen; hence the term obliterating. The media and adventitia are also infiltrated with small cells. This form of endarteritis described by Heubner is not, however, characteristic of syphilis, and its presence alone in an artery could not be considered pathognomonic. If, however, there are gummata in other parts, or if the condition about to be described exists in adjacent arteries, the process may be regarded as syphilitic.

(*b*) *Gummatous Peri-arteritis.*—With or without involvement of the intima, nodular gummata may develop in the adventitia of the artery, producing globular or ovoid swellings, which may attain considerable size. They are not infrequently seen in the cerebral arteries, which seem to be specially prone to this affection. This form is specific and distinctive of syphilis. The disease usually affects the smaller vessels and may be found in the coronary arteries, and particularly in those of the brain.

F. **Renal Syphilis.**—Gummata occasionally develop in the kidneys, particularly in cases in which there is extensive gummatous hepatitis. They are rarely numerous, and occasionally lead to scattered cicatrices. Clinically the affection is not recognizable.

G. **Syphilitic Orchitis.**—This affection is of special significance to the physician, as its detection frequently clinches the diagnosis in obscure internal disorders. Syphilis occurs in the testes in two forms:

(*a*) The *gummatous growth*, forming an indurated mass or group of masses in the substance of the organ, and sometimes difficult to distinguish from tuberculous disease. The area of induration is harder and it affects the body of the testes, while tubercle more commonly involves the epididymis. It rarely tends to invade the skin, or to break down, soften, and suppurate, and is usually painless.

(*b*) There is an *interstitial orchitis* regarded as syphilitic, which leads to fibroid induration of the gland and gradually to atrophy. It is a slow, progressive change, coming on without pain, usually involving one organ more than another.

General Diagnosis of Syphilis.—There is seldom any doubt concerning the existence of syphilitic lesions. The negative statements of the patient must be taken with extreme caution, as persons will lie deliberately with reference to primary infection, when it is in their best interest to make a straightforward truthful statement. It is to be remembered that syphilis is common in the community, and there are probably more families with a luetic than with a tuberculous taint. It is possible that the primary sore may have been of trifling extent, or urethral and masked by a gonorrhœa, and the patient may not have had severe secondary symptoms, but such instances are extremely rare. Inquiries should be made into the history to ascertain if the patient has had skin rashes, sore throat, or if the hair has fallen out. Careful inspection should

be made of the throat and skin for signs of old lesions. Scars in the groins, the result of buboes, may be taken as positive evidence of infection (Hutchinson). The cicatrices on the legs are often copper-colored, though this cannot be regarded as peculiar to syphilis. The bones should be examined for nodes. In doubtful cases the scar of the primary sore may be found, or there may be signs of atrophy or of hardening of the testes. In women, special stress has been laid upon the occurrence of frequent miscarriages, which, in connection with other circumstances, are always suggestive.

In the congenital disease, the occurrence within the first three months of snuffles and skin rashes is conclusive. Later, the characters of the syphilitic facies, already referred to, often give a clew to the nature of some obscure visceral lesion. Other distinctive features are the symmetrical development of nodes on the bones and the interstitial keratitis.

In doubtful cases much stress is laid by some writers upon the therapeutic test, by placing the patient upon antisyphilitic treatment. In the case of an obstinate skin rash of doubtful character, which has resisted all other forms of medication, this has much greater weight than in obscure visceral lesions. I have on several occasions known such marked improvement to follow large doses of iodide of potassium that the diagnosis of syphilitic lesion was greatly strengthened, but the subsequent course and the post-mortem have shown that the disease was not syphilis.

Prophylaxis.—Irregular intercourse has existed from the beginning of recorded history, and unless man's nature wholly changes—and of this we can have no hope—will continue. Resisting all attempts at solution, the social evil remains the great blot upon our civilization, and inextricably blended with it is the question of the prevention of syphilis. Two measures are available—the one personal, the other administrative.

Personal purity is the prophylaxis which we, as physicians, are especially bound to advocate. Continence may be a hard condition (to some harder than to others), but it can be borne, and it is our duty to urge this lesson upon young and old who seek our advice in matters sexual. Certainly it is better, as St. Paul says, to marry than to burn, but if the former is not feasible there are other altars than those of Venus upon which a young man may light fires. He may practise at least two of the five means by which, as the physician Rondibilis counselled Panurge, carnal concupiscence may be cooled and quelled—hard work of body and hard work of mind. Idleness is the mother of lechery; and a young man will find that absorption in any pursuit will do much to cool passions which, though natural and proper, cannot in the exigencies of our civilization always obtain natural and proper gratification.

The second measure is a rigid and systematic regulation of prostitution. The state accepts the responsibility of guarding citizens against small-pox or cholera, but in dealing with syphilis the problem has been

too complex and has hitherto baffled solution. On the one hand, inspection, segregation, and regulation are difficult if not impossible to carry out; on the other hand, public sentiment, in Anglo-Saxon communities at least, is as yet bitterly opposed to this plan. While this feeling, though unreasonable, as I think, is entitled to consideration, the choice lies between two evils—licensing, even imperfectly carried out, or wide-spread disease and misery. If the offender bore the cross alone, I would say, forbear; but the physician behind the scenes knows that in countless instances syphilis has wrought havoc among innocent mothers and helpless infants, often entailing life-long suffering. It is for them he advocates protective measures.

Treatment.—We must admit that various organizations react very differently to the poison of syphilis. There are individuals who, although receiving brief and unsatisfactory treatment, display for years no traces of the disease. On the other hand, there are persons thoroughly and systematically treated from the outset who display from time to time well-marked indications of the disease. Certainly there are grounds for the opinion that persons who have suffered very slightly from secondary symptoms are more prone to have the severer visceral lesions of the later stage.

When we consider that syphilis is one of the most amenable of all diseases to treatment, it is lamentable that the later stages which come under the charge of the physician are so common. This results, in great part, from carelessness of the patient, who, wearied with treatment, cannot understand why he should continue to take medicine after all the symptoms have disappeared; but, in part, the profession also is to blame for not insisting more urgently in every instance that acquired syphilis is not cured in a few months, but takes at least two years, during which time the patient should be under careful supervision. The treatment of the disease is now practically narrowed to the use of two remedies, justly termed specifics—namely, mercury and iodide of potassium. The former is of special service in the secondary, the latter in the tertiary manifestations of the disease; but they are often combined with advantage.

Mercury may be given by the mouth in the form of gray powder, the hydrargyrum cum cretâ, which Hutchinson recommends to be given in pills, one-grain dose with a grain of Dover's powder. One pill from four to six times a day will usually suffice. I warmly endorse the excellent results which are obtained by this method, under which the patient often gains rapidly in weight, and the general health improves remarkably. It may be continued for months without any ill effects. Other forms given by the mouth are the pilules of the biniodide (gr. $\frac{1}{16}$), or of the protiodide (gr. $\frac{1}{8}$), three times a day.

Inunction is a still more effective means. A drachm of the ordinary mercurial ointment is thoroughly rubbed into the skin every evening for six days; on the seventh a warm bath is taken, and on the eighth the mer-

curial course is resumed. At least half an hour should be given to each inunction. It is well to apply it at different places on successive days. The sides of the chest and abdomen and the inner surfaces of the arms and thighs are the best positions.

The mercury may be given by direct injection into the muscles. If proper precautions are taken in sterilizing the syringe, and if the injections are made into the muscles, not into the subcutaneous tissue, abscesses rarely result. One third of a grain of the bichloride in twenty drops of water may be injected once a week, or from one to two grains of calomel in glycerin (20 minims).

Still another method, greatly in vogue in certain parts of the Continent and in institutions, is fumigation. It may be carried out effectively by means of Lee's lamp. The patient sits on a chair wrapped in blankets, with the head exposed. The calomel is volatilized and deposited with the vapor on the patient's skin. The process lasts about twenty minutes, and the patient goes to bed wrapped in blankets without washing or drying the skin. A patient under mercurial treatment should avoid stimulants and live a regular life, not necessarily abstaining from business. Green vegetables and fruit should not be taken. Salivation is to be avoided. The teeth should be cleansed twice a day, and if the gums become tender, the breath fetid, or the tongue swollen and indented, the drug should be suspended for a week or ten days.

In congenital syphilis the treatment of cases born with bullæ and other signs of the disease is not satisfactory, and the infants usually die within a few days or weeks. The child should be nursed by the mother alone, or, if this is not feasible, should be hand-fed, but under no circumstances should a wet-nurse be employed. The child is most rapidly and thoroughly brought under the influence of the drug by inunction. The mercurial ointment may be smeared on the flannel roller. This is not a very cleanly method, and sometimes rouses the suspicion of the mother. It is preferable to give the drug by the mouth, in the form of gray powder, half a grain three times a day. In the late manifestations associated with bone lesions, the combination of mercury and iodide of potassium is most suitable and is well given in the form of Gilbert's syrup, which consists of the biniodide of mercury (gr. j), of potassium iodide (℥ ss.), and water (℥ ij). Of this a dose for a child under three is from five to ten drops three times a day, gradually increased. Under these measures, the cases of congenital syphilis usually improve with great rapidity. The medication should be continued at intervals for many months, and it is well to watch these patients carefully during the period of second dentition and at puberty, and if necessary to place them on specific treatment.

In the treatment of the visceral lesions of syphilis, which come more distinctly within the province of the physician, iodide of potassium is of equal or even greater value than mercury. Under its use ulcers rapidly heal, gummatous tumors melt away, and we have an illustration of a spe-

cific action only equalled by that of mercury in the secondary stages, by iron in certain forms of anæmia, and by quinine in malaria. It is as a rule well borne in an initial dose of ten grains, or ten minims of the saturated solution; given in milk the patient does not notice the taste. It should be gradually increased to thirty or more grains three times a day. In syphilis of the nervous system it may be used in still larger doses. Seguin, who has specially insisted upon the advantage of this plan, urges that the drug should be pushed, as good effects are not obtained with the moderate doses.

When syphilitic hepatitis is suspected the combination of mercury and iodide of potassium is most satisfactory. If there is ascites, Addison's or Niemeyer's pill (as it is often called) of calomel, digitalis, and squills will be found very useful. A patient of mine with recurring ascites, on whom paracentesis was repeatedly performed and who had an enlarged and irregular liver, took this pill for more than a year with occasional intermissions, and ultimately there was a complete disappearance of the dropsy and an extraordinary reduction in the volume of the liver. Occasionally the iodide of sodium is more satisfactory than the iodide of potassium. It is less depressing and agrees better with the stomach. Many patients possess a remarkable idiosyncrasy to the iodide, but as a rule it is well borne. Severe coryza with salivation, and œdema about the eyelids, are its most common disagreeable effects. Skin eruptions also are frequent. I have known patients unable to take more than from twenty to thirty grains without suffering from an erythematous rash; much more common is the acne eruption. Occasionally an urticarial rash may develop with spots of purpura. Some of these iodide eruptions may closely resemble syphilis. Hutchinson has reported instances in which they have proved fatal.

Upon the question of syphilis and marriage the family physician is often called to decide. He should insist upon the necessity of two full years elapsing between the date of infection and the contracting of marriage. This, it should be borne in mind, is the earliest possible limit, and there should be at least a year of complete immunity from all manifestations of the disease.

In relation to life insurance, an individual with syphilis can not be regarded as a first-class risk unless he can furnish evidence of prolonged and thorough treatment and of immunity for two or three years from all manifestations. Even then, when we consider the extraordinary frequency of the cerebral and other complications in persons who have had this disease and who may even have undergone thorough treatment, the risk to the company is certainly increased.

XXVI. TUBERCULOSIS.

I. General Etiology and Morbid Anatomy.

Definition.—An infective disease, caused by the *bacillus tuberculosis*, the lesions of which are characterized by nodular bodies called tubercles or diffuse infiltrations of tuberculous tissue which undergo caseation or sclerosis and may finally ulcerate, or in some situations calcify.

Etiology.—1. **Zoölogical Distribution.**—Tuberculosis is one of the most wide-spread of maladies.

In cold-blooded animals it is rare, owing doubtless to temperature conditions unfavorable to the development of the bacillus. Among reptiles in confinement it is, however, occasionally seen (Sibley). In fowls it is an extremely common disease, but recent facts indicate that there are differences in avian tuberculosis sufficient to warrant its separation from the ordinary form.

Among domestic animals tuberculosis is widely but unevenly distributed. Among ruminants, bovines are chiefly affected. The statistics of the Berlin abattoir show that in the years 1887–'88 tuberculosis was found in 4,300 cattle. In this country it is extremely difficult to get satisfactory statistics of the prevalence of the disease. At the meeting of the United States Veterinary Medical Association in 1889, it was stated in a resolution that from ten to fifteen per cent of the dairy stock of the Eastern States was tuberculous.

In Baltimore, A. W. Clement, United States veterinary inspector, informs me that of 5,297 cattle slaughtered in Maryland only 159 were tuberculous.

In sheep the disease is very rare. In pigs it is common, but not so common in this country as in Europe. In the inspection of one thousand hogs, which was made by A. W. Clement and myself in Montreal in 1880, tuberculosis was seen only once or twice. At the Berlin abattoir in 1887–'88 there were 6,393 pigs affected with the disease.

Horses are rarely attacked. Dogs and cats are not prone to the disease, but cases are described in which infection of pet animals has taken place from phthisical masters. Among the semi-domestic animals, such as the rabbit and guinea-pig, the disease under natural conditions is rare, although these animals, particularly the latter, are extremely susceptible to the disease when inoculated. Among apes and monkeys in the wild state, tuberculosis is unknown, but in confinement it is the most formidable disease with which they have to contend.

The important etiological fact in connection with tuberculosis in animals is the wide-spread occurrence of the disease in bovines, from which class we derive nearly all the milk and a very large proportion of the meat used for food.

2. **Geographical Distribution.**—The disease exists in all countries. It

prevails more in the large cities and wherever the population is massed together Thus, while the general death-rate from it is three per thousand, that of Vienna is 7·7, and of Munich and Glasgow four per thousand. Hirsch, from whose classical work these facts are taken, thinks that geographical position has less influence than has been supposed. Italy and England suffer alike, and the disease is very prevalent in the West Indies and the South Sea islands. Toward the poles it is rare. It is a common disease in Canada, and prevails extensively among the French Canadians and the English. Altitude is a more potent factor than latitude. In the high regions of the Alps and Andes, and in the central plateau of Mexico the disease is very rare. Mountainous countries, such as Switzerland, have a very low death-rate from tuberculosis.

3. **Race.**—No race is immune. The Indians of this continent are very prone to the disease. Matthews, whose experience with the native race is large, states that the disease is on the increase among them. He quotes the ratio from the United States census, 1880, as white 166, negroes 186, Indians 286. The death-rate in the older reservations, as in New York, is three times as great as in Dakota. In the Blood Indian Reserve of the Canadian Northwest Territories, Surgeon Kennedy (N. W. M. P.) has given me the figures for six years. In a population of about 2,000 there were 127 deaths from pulmonary consumption, twenty-three per cent of the total rate. This does not include deaths from "diseases of infancy." This enormous death-rate, it is to be remembered, occurs in a tribe occupying one of the finest climates of the world among the foot-hills of the Rocky Mountains, a region in which consumption is extremely rare among the white population, and in which cases of tuberculosis from the eastern provinces do remarkably well.

The negro race is very susceptible to tuberculosis, more particularly the glandular and osseous forms. Of the 427 cases of pulmonary tuberculosis at the Johns Hopkins Hospital for the two years ending June 1, 1891, there were 41 cases in the colored—i. e., about 1:10. The ratio of colored to white of all patients in the wards has been 1 to 7.

4. **The Bacillus Tuberculosis.**—The history of the discovery of the bacillus presents many points of interest. Confidently expected by such observers as Villemin, Chauveau, Cohnheim, and others, and claimed to have been demonstrated by many, notably by Klebs and Aufrecht, it remained for Koch to demonstrate its existence and its invariable association with the disease. The investigations which he had previously made upon anthrax and experimental traumatic infections, by perfecting the methods of research, paved the way for this brilliant discovery. His preliminary article * and his more elaborate later work † should be carefully studied by any one who wishes to appreciate the value of scientific methods. It forms

* Berliner klinische Wochenschrift, 1882.

† Mittheilungen a. d. k. Gesundheitsamte, Bd. 2.

one of the most masterly demonstrations of modern medicine. Its thoroughness appears in the fact that in the nine years which have elapsed since its announcement the innumerable workers at the subject have not, so far as I know, added a solitary essential fact to those presented by Koch.

Morphological Characters.—The tubercle bacillus is a short, fine rod, often slightly bent or curved, and has an average length of nearly half the diameter of a red blood-corpuscle (3 to 4 μ). When stained it often presents a beaded appearance, which some have attributed to the presence of spores.

With the basic aniline dyes it stains slowly, except at the body temperature, but retains the dye after treatment with acids—a characteristic which separates it from all other known forms of bacteria, with the exception of the bacillus of leprosy.

Modes of Growth.—It grows on blood-serum, glycerin-agar, or on potato—most readily on the former. The cultures must be kept at blood-heat. They grow slowly, and do not appear until about the end of the second week. The colonies form thin, grayish-white, dry, scale-like masses on the surface of the culture medium. Successive inoculations may be made from the cultures, and at the end of an indefinite series material from one of them inoculated into a guinea-pig will produce tuberculosis.

Products of the Growth.—Little is yet known of the chemical characters of the materials which result from the growth of the tubercle bacilli. Koch's tuberculin is stated to be a glycerin extract of the cultures. Crookshank and Herroun have separated an albumose and a ptomaine.

Distribution of the Bacilli.—The bacilli are found in all tuberculous lesions; in some in great abundance, in others sparsely. They are particularly numerous in actively developing tubercles, but in the chronic tuberculous processes of lymph-glands and of the joints they are scanty. When a tuberculous focus communicates with a vein or with lymph-vessels, the bacilli may be spread widely throughout the body. In old lesions they may not be found in the sections, and the demonstration of the true nature may be possible only by culture or inoculation.

The Bacilli outside the Body.—Patients with advanced pulmonary tuberculosis throw off in the expectoration countless millions of the bacilli daily. Some idea of the extraordinary numbers may be gained from the studies of Nuttall.* From a patient in my ward, with moderately advanced disease, the amount of whose expectoration was from seventy to a hundred and thirty cubic centimetres daily, he estimated by his method that there were in sixteen counts, between January 10th and March 1st, from one and a half to four and a third billions of bacilli thrown off in the twenty-four hours. These figures emphasize the danger associated with phthisical sputa unless most carefully dealt with. When expectorated and allowed to dry, the sputum rapidly becomes dust, and is distributed far and wide.

* Johns Hopkins Hospital Bulletin, May, 1891.

The observations made by Cornet under Koch's supervision are in this connection most instructive. He collected the dust from the walls and bedsteads of various localities, and determined its virulence or innocuousness by inoculation into susceptible animals. Material was gathered from twenty-one wards of seven hospitals, three asylums, two prisons, from the surroundings of sixty-two phthisical patients in private practice, and from twenty-nine other localities in which tuberculous patients were only transient frequenters (out-patient departments, streets, etc.). Of one hundred and eighteen dust samples from hospital wards or the rooms of phthisical patients, forty were infective and produced tuberculosis. Negative results were obtained with the twenty-nine dust samples from the localities occasionally occupied by consumptives. Virulent bacilli were obtained from the dust of the walls of fifteen out of twenty-one medical wards. It is interesting to note that in two wards with many phthisical patients the results were negative, indicating that the dust in such regions is not necessarily infective. The infectiousness of the medical and surgical divisions of a hospital is in the proportion of 76·6 to 12·5. In a room in which a tuberculous woman had lived, the dust from the wall in the neighborhood of the bed was infective six weeks after her death. No bacilli were found in the dust of an inhalation-chamber for consumptives.

The tubercle bacillus is thus a wide-spread organism in regions frequented by phthisical patients.

5. **Modes of Infection.**—(*a*) *Hereditary Transmission.*—In extremely rare instances the disease is congenital. A few undoubted cases have been reported in man and in the calf. The rarity with which it occurs may be gathered from the fact that of 15,400 calves killed at the Berlin abattoir there were only four instances of tuberculosis. Cases of congenital tuberculosis in man have occasionally been described.

Baumgarten holds that in many cases the virus is transmitted, but the disease does not appear until some time after birth. He bases this opinion upon the following facts:

The great frequency of tuberculosis in sucklings. Thus, in 16,581 autopsies on sucklings, Frobelius found 416 with tuberculous lesions. In 219 cases of tuberculosis in children under two, from Parrot's clinic, there were 23 under three months, and a total of 111 under one year. It seems probable that in many of these cases the virus itself was transmitted.

The common occurrence of tuberculosis in the bones and in the joints of children, regions to which it seems unlikely that the bacilli would be conveyed in accidental infection. To make this objection valid we should require a series of cases of bone tuberculosis in children in which examination showed the lymph portals of the bronchi and the mesentery to be free from disease. He regards the late manifestation as analogous to the *syphilis hereditaria tarda*, and suggests that the growth of the germs is,

as a rule, restrained or held in check by the actively developing tissues of the child.

Tuberculosis unquestionably may be inherited, but in what way and how often are unsettled problems. Congenital disease is extremely rare, but there is no inherent improbability in a prolonged latency of the virus. That it may be present without the existence of actual tubercles is indicated by an experiment of Birch-Hirschfeld, who found that portions of the viscera of a fœtus born of a phthisical mother, though not itself tuberculous, were infective to guinea-pigs.

In any series of cases of pulmonary tuberculosis there is a suspicious number in which the ascendants have also been tuberculous. Thus, in 427 cases at the Johns Hopkins Hospital there were 53 in which the mother was affected, 52 in which the father had tuberculosis, and 105 in which brother or sister had had the disease. The estimates by various authors range from 10 per cent (Louis), 25 per cent (Walshe), to even 50 per cent. Fagge very justly remarks that it is impossible to draw a line between hereditary and accidental tuberculosis, and naturally the children of an affected parent are more liable to accidental contamination. Maternal is very much more common than paternal inheritance. A family tree, such as is here given, of six generations tells its own tale. It is interesting to note the almost constant transmission through the mother.

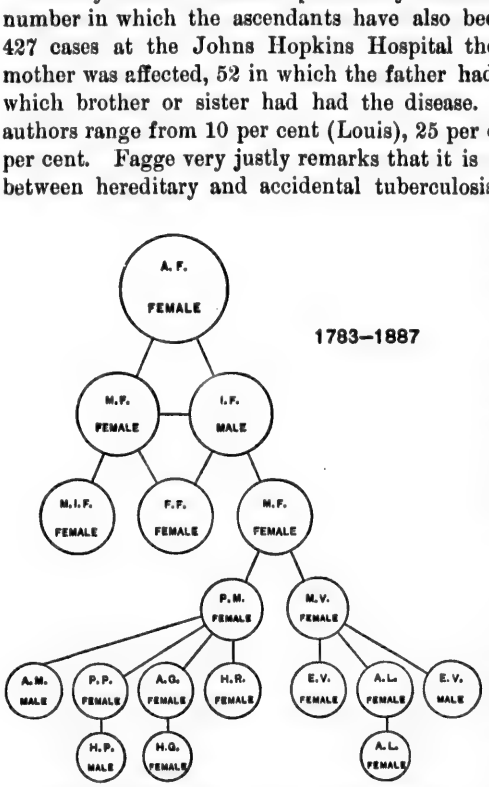

CHART XII.—Heredity in pulmonary tuberculosis.

(*b*) *Inoculation.*—The infective nature of tuberculosis was first demonstrated by Villemin, who showed conclusively in 1865 that it could be transmitted to animals by inoculation. The question was hotly contested, and Villemin's observations were confirmed by Simon, Andrew Clark, and others, but Burdon Sanderson, Wilson Fox, and others held that the disease could be transmitted by non-tuberculous materials. The beautiful experiments of Cohnheim and Salamonson, who produced tuberculosis in the eyes of guinea-pigs and rabbits by inoculating fresh tubercle into the ante-

rior chamber, confirmed and extended Villemin's original observations and paved the way for the reception of Koch's announcement. It is now universally conceded that *only* tuberculous matter can produce, when inoculated, tuberculosis. In man tuberculosis is not often transmitted by inoculation, and when it does occur the disease usually remains local. This mode of infection is seen in persons whose occupation brings them in contact with dead bodies or animal products. Demonstrators of morbid anatomy, butchers, and handlers of hides are subject to a local tubercle of the skin, which forms a reddened mass of granulation tissue, usually capping the dorsal surfaces of the hands or fingers. This is the so-called post-mortem wart, the *verruca necrogenica* of Wilks. The demonstration of its nature is shown by the presence of tubercle bacilli, and by inoculation experiments in animals.

The statement that Laennec contracted phthisis from this source is probably false, since he did not die until twenty years after the inoculation and in the interval presented no manifestations. The possibility, however, of general infection must be borne in mind. Gerber reports that after accidental inoculation of the hand from a case of phthisis he had for months a "Leichen-tubercle," which was excised. Shortly afterward the lymph-glands of the axilla became enlarged and painful, and when removed showed characteristic tuberculous changes, with bacilli.

In the performance of the rite of circumcision children have been accidentally inoculated. Infection in these cases is probably always associated with disease in the operator, and occurs in connection with the habit of cleansing the wound by suction.

Other means of inoculation have been described: as the wearing of ear-rings, washing the clothes of phthisical patients, the bite of a tuberculous subject, or inoculation from a cut by a broken spit-glass of a consumptive; and Czerny has reported two cases of infection by transplantation of skin.

It has been urged by the opponents of vaccination that tuberculosis, as well as syphilis, may be thus conveyed, but of this there is no evidence, and the lymph from the vesicles of revaccinated consumptives has been shown by many observers to be non-infective. It may be said, on the whole, that inoculation in man plays a trifling *rôle* in the transmission of tuberculosis.

(*c*) *Infection through the Air.*—It has been fully proved that the expired air of tuberculous patients is not infective. On the other hand, the virus is contained in enormous amounts in the sputum, which, when dried, is soon widely disseminated in the form of dust, and unless carefully sterilized constitutes a great medium of transmission. A belief in the contagiousness of pulmonary tuberculosis has existed from the days of the early Greek physicians, and has persisted among the Latin races.

The investigations of Cornet afford conclusive proof that the dust of a

room or other locality frequented by patients with pulmonary tuberculosis is infective. The bacilli are attached to fine particles of dust and in this way gain entrance to the system through the lungs. The following are some of the facts in favor of this view:

(1) Primary tuberculous lesions are in a majority of all cases connected with the respiratory system. The frequency with which foci are met with in the lungs and in the bronchial glands is extraordinary, and the statistics of the Paris morgue show that a considerable proportion of all persons dying of accident or by suicide present evidences of the disease in these parts. The post-mortem statistics of hospitals show the same wide-spread prevalence of infection through the air-passages. Biggs reports that more than 60 per cent of his post-mortems showed lesions of pulmonary tuberculosis. In one hundred and twenty-five post-mortems at the Foundling Hospital, New York, the bronchial glands were tuberculous in every case. In adults the bronchial glands may be infected while the individual is in good health. H. P. Loomis found in eight of thirty cases in which there were no signs of old or recent tuberculous lesions that the bronchial glands were infective to rabbits.

(2) The greater prevalence of tuberculosis in institutions in which the residents are confined and restricted in the matter of fresh air and a free open life—conditions which would favor, on the one hand, the presence of the bacilli in the atmosphere, and, on the other, lower the vital resistance of the individual. The investigations of Cornet upon the death-rate from consumption among certain religious orders devoted to nursing give some striking facts in illustration of this. In a review of thirty-eight cloisters, embracing the average number of 4,028 residents, among 2,099 deaths in the course of twenty-five years, 1,320 (62·88 per cent) were from tuberculosis. In some cloisters more than three fourths of the deaths are from this disease, and the mortality in all the residents, up to the fortieth year, is greatly above the average, the increase being due entirely to the prevalence of tuberculosis. It has been stated that nurses are not more prone to the disease than other individuals, but Cornet says that of a hundred nurses deceased, sixty-three died of tuberculosis. The more perfect the prophylaxis and hygienic arrangements of an asylum or institution, the lower the mortality from tuberculosis. The mortality in prisons has been shown by Baer to be four times as great as outside. The death-rate from phthisis is estimated at 15 per cent of the total mortality, while in prisons it constitutes from 40 to 50 per cent, and in some countries, as Austria, over 60 per cent. Flick has studied the distribution of the deaths from tuberculosis in a single city ward in Philadelphia for twenty-five years. His researches go far to show that it is a house disease. About 33 per cent of infected houses have had more than one case. Less than one third of the houses of the ward became infected with tuberculosis during the twenty-five years prior to 1888. Yet more than one half of the deaths from this disease during the year 1888 occurred in those in-

fected houses. There are, however, opposing facts. The statistics of the Brompton Consumption Hospital show that doctors, nurses, and attendants are rarely attacked. Dettweiler claims that no case of tuberculosis has been contracted among his nurses or attendants at Falkenstein. The whole question has recently been thoroughly reviewed by Heron,* in whose work will also be found a list of cases of infection (prepared by Koch) reported between 1867 and 1889.

(3) Special danger exists when the contact is very intimate, such, for instance, as between man and wife. On this point much difference of opinion exists, but the figures seem to indicate that under these circumstances the husband or wife is much more liable subsequently to die of consumption. Of 427 cases of pulmonary tuberculosis at the Johns Hopkins Hospital, in 25 either husband or wife had been affected with it or had died of tuberculosis. In response to a question as to contagion, asked by the Collective Investigation Committee of the British Medical Association, there were 261 replies in the affirmative, among which were 158 cases of supposed contagion through marriage. Weber's cases are of special interest. One of his patients lost four wives in succession, one lost three, and four lost two each.

(*d*) *Infection by Milk.*—The milk of an animal suffering from tuberculosis may contain the virus, and is capable of communicating the disease, as shown by Gerlach, Bang, Bollinger, and others. Striking illustrations of this are sometimes afforded in the lower animals. The pigs, for instance, of a tuberculous sow have been shown to present intestinal tuberculosis of the most exquisite form. Of late years the experimental proof has been entirely conclusive. It was formerly thought that the cow must present tuberculous disease of the udder, but Ernst has shown that the bacilli may be present and the milk be infective in a large proportion of cases in which there is no tuberculous mammitis; an observation made also by Hirschberger and others. This author states the interesting fact that an owner of a herd known to be tuberculous withdrew the milk from market and used it without boiling to fatten his pigs, which, almost without exception, became tuberculous, so that the whole stock had to be slaughtered. There is no reason to believe that young children or even adults are less susceptible to the virus than calves or pigs, so that the danger of the disease from this source is real and serious. The great frequency of intestinal and mesenteric tuberculosis in children no doubt finds here its explanation. As noted in Woodhead's analysis of one hundred and twenty-seven cases of fatal tuberculosis in children, the mesenteric glands were involved in one hundred.

(*e*) *Infection by Meat.*—The meat of tuberculous animals is not necessarily infective. The results of experiments with the flesh of cows are not in accord. This mode of infection probably plays a minor *rôle* in the

* Evidences of the Communicability of Consumption, London, 1890.

etiology of human tuberculosis, as usually the flesh is thoroughly cooked before eating. The possibility, however, must be borne in mind, and it would certainly be safer in the interests of a community to confiscate the carcasses of all tuberculous animals. Experiments in Bollinger's laboratory show that the flesh of tuberculous subjects is very infective to guinea-pigs.

6. **Conditions influencing Infection.**—(*a*) *Constitutional Peculiarities.*—It was formerly thought that individuals of a certain habit of body, and of a certain physiognomy, the *habitus phthisicus*, were specially prone to tuberculous disease; but few now regard the so-called tuberculous or scrofulous diathesis as more than an indication of a certain type of conformation, in which the tissues are more vulnerable and less capable of resisting infection. In many instances Cohnheim is unquestionably correct in stating that the so-called phthisical *habit* is not an indication of a tendency to, but actually of the existence of, tuberculosis. The belief in a special phthisical frame has existed in the profession from the days of Hippocrates, who says, "The form of body peculiar to subjects of phthisical complaints was the smooth, the whitish, that resembling the lentil; the reddish, the blue-eyed, the leuco-phlegmatic, and that with the scapulæ having the appearance of wings." Galen also wrote upon this type of chest as specially characteristic of the disease. Certainly the long, narrow, flat chest with depressed sternum is most commonly seen in tuberculous persons, but how common it is also to meet with patients who have well-formed, well-built chests, with wide costal angle and good pulmonary expansion! The investigations of Beneke with reference to the formation of the viscera in the subjects of phthisis are very interesting. His measurements indicate that the heart is relatively small, the arteries are proportionately narrow, and the pulmonary artery is relatively wider than the aorta. This point, he suggests, would lead to increase in the blood-pressure in the lungs and favor catarrh. The lung volume he found to be relatively greater in those affected with phthisis.

Galton and Mahomed made observations upon the composite portraiture of phthisis. In 442 patients they separated two types of face; one ovoid and narrow, the other broad and coarse featured. This corresponds in an interesting way to the diathetic states formerly recognized—namely, the tuberculous, with thin skin, bright eyes, oval face, and long, thin bones; and the scrofulous, with thick lips and nose, opaque skin, large thick bones, and heavy figure. These conditions, on which so much stress was formerly laid, indicate, as Fagge states, nothing more than delicacy of constitution, incomplete growth, and imperfect development.

(*b*) *Influence of Age.*—Tuberculosis occurs at all periods of life, in the suckling as well as in the octogenarian. The distribution of the lesions varies greatly at different ages. In the first decade the lymphatic glands, bones, and meninges are much more frequently affected than at subsequent periods. Meningeal tuberculosis is most common between the third and eighth years.

The mesenteric glands are specially prone to be involved in young children, as before mentioned. Of 127 cases of tuberculosis in children, Woodhead found these bodies affected in 100 instances, in 14 of which there were no tubercles in other parts of the body. The majority of these cases occur between the first and fifth years. The bronchial glands are still more frequently involved, and of 125 cases at the New York Foundling Hospital in every one were these structures the seat of more or less extensive tuberculosis.

In adults the lungs usually contain tubercle when it is present in the body (Louis' law).

(*c*) *Soil and locality* are held by many to have an important influence in tuberculosis. The observations of H. I. Bowditch in this country, and of Buchanan in England, show that pulmonary tuberculosis is more prevalent in damp, ill-drained districts; but this increased incidence is most probably associated with a heightened vulnerability due to an increased liability to catarrhal affections of all kinds.

(*d*) *Local Conditions influencing Infection.*—These are doubtless of the highest importance, and second only to the constitutional vulnerability. Among the more important may be mentioned:

Catarrhal Inflammation.—This probably acts by lowering the resistance, or, in modern parlance, reducing the activity of the phagocytes and allowing the bacilli to pass the portals. The liability of infection in the cervical and bronchial glands in children is probably associated with the common occurrence of catarrhal processes in the throat and bronchi.

The influence of bronchial catarrh in pulmonary tuberculosis is all-important. How often is it said that the disease has started in a neglected cold, which means, in other words, that the bronchial catarrh has enfeebled the power of tissue resistance, or produced conditions favorable to the growth and development of the bacilli!

An important part in the etiology of tuberculous processes is played by *trauma*. Surgeons have for years laid great stress upon this association, but the relation, though universally recognized, is by no means easy of explanation. Bacteriological experiments, however, indicate that in tissues which have been injured organisms, which would in health have been readily and rapidly destroyed by the action of the normal juices or cells, under these altered circumstances grow rapidly and develop. Probably in the case of tuberculosis following trauma the injured part is for a time a *locus minoris resistentiæ*, and if bacilli are present they may by it receive a stimulus to growth, or under the altered conditions be capable of multiplying. Not only in arthritis but in pulmonary tuberculosis traumatism may play a part. The question has been thoroughly studied by Mendelsohn,* who reports nine cases in which, without fracture of the

* Zeitschrift f. klin. Medicin, Bd. 10.

rib or laceration of the lung, tuberculosis developed shortly after contusion of the chest.

The production of general tuberculosis is sometimes favored by *operation* upon tuberculous lesions. Surgeons have long known that resection of a strumous joint is occasionally followed by acute tuberculosis. The question has been carefully studied by Wartmann,* who gives statistics of 837 resections. Of these, 225 ended fatally, 26 with acute tuberculosis, the outbreak of which was directly associated with operation.

The acute miliary tuberculosis which, as Litten has shown, occasionally follows the aspiration of the effusion in tuberculous pleurisy, may come under this division.

The constant inhalation of *impure air* in occupations associated with a very dusty atmosphere renders the lungs less capable of resisting infection. The pulmonary affection of stone-cutters and coal-miners, though non-tuberculous at the outset and often a simple chronic interstitial pneumonia, is ultimately in a large proportion of the cases tuberculous. In manufactories metallic seems more hurtful than mineral dust. Peterson† quotes the incidence of pulmonary tuberculosis among the trades as follows: Glass-workers, 80 per cent; needle-sharpeners, 70; file-cutters, 62; and stone-cutters, 60. And, lastly, circumstances which temporarily lower the nutrition, as the *specific fevers* render the tissues more susceptible. In this way alone can we explain the frequent onset of tuberculosis after an exhausting illness. Fevers, such as measles and whooping-cough, which are associated with bronchial catarrh, are more prone than others to be followed by tuberculosis. This is often only the blazing of a smouldering fire.

General Morbid Anatomy and Histology of Tuberculous Lesions.

(1) **Distribution of the Tubercles in the Body.**—The organs of the body are variously affected by tuberculosis. In adults, the lungs may be regarded as the seat of election; in children, the lymph-glands, bones, and joints. In 1,000 autopsies there were 275 cases with tuberculous lesions. With but two or three exceptions the lungs were affected. The distribution in the other organs was as follows: Pericardium, 7; peritonæum, 36; brain, 31; spleen, 23; liver, 12; kidneys, 32; intestines, 65; heart, 4; and generative organs, 8.

The tuberculosis which comes under the care of the surgeon has a different distribution, as shown by the following figures from the Wurzburg clinic: Among 8,873 patients there were 1,287 tuberculous, with the following distribution of lesions: Bones and joints, 1,037; lymph-glands, 196; skin and connective tissues, 77; mucous membranes, 10; genito-urinary organs, 20.

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* Deutsche Zeitschrift f. Chirurgie, Bd. 24.

† Medical News, 1885.

(2) **The Changes produced by the Tubercle Bacilli.**

(*a*) *The Nodular Tubercle.*—The body which we term a "tubercle" *presents in its early formation nothing distinctive or peculiar, either in its components or in their arrangement.* Identical structures are produced by other parasites, such as the actinomyces, and by the strongylus in the lungs of sheep.

The researches of Baumgarten have enabled us to follow in detail all the steps in the development of a tubercle.

These are: ( ) The multiplication of the fixed cells, especially those of connective tissue and the endothelium of the capillaries, and the gradual production from them of rounded, cuboidal, or polygonal bodies with vesicular nuclei—the *epithelioid cells*—inside some of which the bacilli are soon seen.

(β) From the vessels of the infected focus, leucocytes migrate in numbers and form the lymphoid cells which were thought to be so characteristic of tubercle. They do not, however, undergo division.

(γ) A reticulum of fibres is formed by the fibrillation and rarefaction of the connective-tissue matrix. This is most apparent, as a rule, at the margins of the growth.

(δ) In some, but not all, tubercles *giant cells* are formed by an increase in the protoplasm and in the nuclei of an individual cell, or possibly by the fusion of several cells. The giant cells seem to be in inverse ratio to the number and virulence of the bacilli. In lupus, joint tuberculosis, and scrofulous glands, in which the bacilli are scanty, the giant cells are numerous; while in miliary tubercles and all lesions in which the bacilli are abundant the giant cells are few in number.

The bacilli then cause, in the first place, a proliferation of the fixed elements, with the production of epithelioid and giant cells; and, secondly, an inflammatory reaction, associated with exudation of leucocytes. How far the leucocytes attack and destroy the bacilli has not been definitely settled—Metschnikoff claiming, Baumgarten denying, an active phagocytosis.

Once formed, a tubercle undergoes caseation and sclerosis.

Caseation.—At the central part of the growth, owing to the direct action of the bacilli, a process of coagulation necrosis goes on in the cells, which lose their outline, become irregular, no longer take stains, and are finally converted into a homogeneous, structureless substance. Proceeding from the centre outward, the tubercle may be gradually converted into a yellowish-gray body, in which, however, the bacilli are still abundant. No blood-vessels are found in them. Aggregated together these form the cheesy masses so common in tuberculosis, which may undergo (*a*) softening; (*b*) fibroid limitation (encapsulation); (*c*) calcification.

Sclerosis.—With the necrosis of the cell elements at the centre of the tubercle, hyaline transformation proceeds, together with great increase in the fibroid elements; so that the tubercle is converted into a firm, hard

structure. Often the change is rather of a fibro-caseous nature; but the sclerosis predominates. In some situations, as the peritonæum, this seems to be the natural transformation of tubercle, and it is by no means rare in the lungs.

In all tubercles two processes go on: the one—caseation—destructive and dangerous; and the other—sclerosis—conservative and healing. The ultimate result in a given case depends upon the capabilities of the body to restrict and limit the growth of the bacilli. There are tissue-soils in which the bacilli are, in all probability, killed at once—*the seed has fallen by the wayside.* There are others in which a lodgment is gained and more or less damage done, but finally the day is with the conservative, protecting forces—*the seed has fallen upon stony ground.* Thirdly, there are tissue-soils in which the bacilli grow luxuriantly, caseation and softening, not limitation and sclerosis, prevail, and the day is with the invaders—*the seed has fallen upon good ground.*

The action of the bacilli injected directly into the blood-vessels illustrates many points in the histology and pathology of tuberculosis. If into the vein of a rabbit a pure culture of the bacilli is injected, the microbes accumulate chiefly in the liver and spleen. The animal dies usually within two weeks, and the organs apparently show no trace of tubercles. Microscopically, in both spleen and liver the young tubercles in process of formation are very numerous, and the process of karyokinesis is seen in the liver-cells. After an injection of a more dilute culture, or one whose virulence has been mitigated by age, instead of dying within a fortnight the animal survives for five or six weeks, by which time the tubercles are apparent in the spleen and liver, and often in the other organs.

(*b*) *The Diffuse Infiltrated Tubercle.*—This is most frequently seen in the lungs. Only a great master like Virchow could have won the profession from a belief in the *unity of phthisis*, which the genius of Laennec had, on anatomical ground, announced. Here and there a teacher, as Wilson Fox, protested, but the heresy prevailed, and we repeated the striking aphorism of Niemeyer, "The greatest evil which can happen to a consumptive is that he should become tuberculous." It was thought that the products of any simple inflammation might become caseous and that ordinary catarrhal pneumonia terminated in phthisis. It was peculiarly fitting that from Germany, in which the dualistic heresy arose, the truth of Laennec's views should receive incontestable proof, in the demonstration by Koch of the etiological unity of all the various processes known as tuberculous and scrofulous.

Infiltrated tubercle results from the fusion of many small foci of infection—so small indeed that they may not be visible to the naked eye, but which histologically are seen to be composed of scattered centres, surrounded by areas in which the air-cells are filled with the products of exudation and of the proliferation of the alveolar epithelium. Under the influence of the bacilli, caseation takes place, usually in small groups of

lobules, occasionally in an entire lobe, or even the greater part of a lung. In the early stage of the process, the tissue has a gray gelatinous appearance, the *gray infiltration* of Laennec. The alveoli contain a sero-fibrinous fluid with cells, and the septa are also infiltrated. These cells accumulate and undergo coagulation necrosis, forming areas of caseation, the *infiltration tuberculeuse jaune* of Laennec, the scrofulous or cheesy pneumonia of later writers. There may also be a diffuse infiltration and caseation without any special foci, a wide-spread tuberculous pneumonia induced by the bacilli.

After all, the two processes are identical. As Baumgarten states: "there is no well-marked difference between miliary tubercle and chronic caseous pneumonia. Speaking histologically, miliary tuberculosis is nothing else than a chronic caseous miliary pneumonia, and chronic caseous pneumonia is nothing but a tuberculosis of the lungs."

(*c*) *Secondary Inflammatory Processes.*—(1) The irritation of the bacilli invariably produces an inflammation which may, as has been described, be limited to exudation of leucocytes and serum, but may also be much more extensive, and varies with varying conditions. We find, for example, about the smaller tubercles in the lungs, pneumonia—either catarrhal or fibrinous, proliferation of the connective-tissue elements in the septa (which also become infiltrated with round cells), and changes in the blood and lymph vessels.

(2) In processes of minor intensity the inflammation is of the slow reactive nature, which results in the production of a cicatricial connective tissue which limits and restricts the development of the tubercles and is the essential conservative element in the disease. It is to be remembered that in chronic pulmonary tuberculosis much of the fibroid tissue which is present is not in any way associated with the action of the bacilli.

(3) Suppuration. Do the bacilli themselves induce suppuration? In so-called cold tuberculous abscess the material is not histologically pus, but a *débris* consisting of broken-down cells and cheesy material. It is moreover sterile—that is, does not contain the usual pus organisms. The products of the tubercle bacilli are probably able to induce suppuration, as in joint and bone tuberculosis pus is frequently produced, although this may be due to a mixed infection. Koch, it will be remembered, states that the "tuberculin" is one of the best agents for the production of experimental suppuration. In tuberculosis of the lungs the suppuration is largely the result of an infection with pus organisms.

II. Acute Tuberculosis.

The truly infective nature of tubercle is best shown in this affection, which is characterized by an eruption of miliary tubercles in various parts of the body. The clinical picture varies with the general or localized distribution of the growths. The tubercles are found upon the pleura and

peritonæum; in the lungs, liver, kidneys, lymph-glands, and spleen; upon the membranes of the brain, occasionally in the choroid coat of the eye, and in the bone-marrow. They may be abundant in some organs and scanty in others. Thus, in the meninges of the brain they may be thickly set, while there are few or none in the abdominal viscera or in the lungs. On the other hand, the lungs may be stuffed with granulations while the meninges of the brain are free. In other cases, again, the distribution is uniform in all the viscera.

The *etiology* has been in part considered, and the only additional statement necessary is that in a great majority of all cases it is an *auto-infection*, arising from a pre-existing tuberculous focus, which may be latent and unsuspected. The following are the most common sources of general infection: Local disease of the lungs, which may be quite limited and unproductive of symptoms; tuberculous affection of the lymph-glands, particularly in children; and tuberculosis of the bones and of the kidneys. Of these sources perhaps the most common are the tracheal and bronchial lymph-glands, which are so often the seat of local tuberculosis. Weigert has shown that in many cases the infection results from the rupture of a caseous pulmonary nodule into a vein, or of a caseous bronchial gland into one of the pulmonary veins. A general infection may, as shown by Ponfick, result from invasion of the thoracic duct by tubercles. With special care the source of infection can usually be discovered at post-mortem examination. The connection between tuberculous lymph-glands and veins has often been demonstrated. In many instances it is impossible to say what determines the sudden and violent onset of the disease. It would seem sometimes as if general rather than local conditions influenced the outbreak. After certain fevers, particularly measles and whooping-cough in children—affections, it is true, which are associated with long-continued bronchitis—miliary tuberculosis is not uncommon. The prostration and constitutional weakness which follow protracted fevers frequently seem in the adult a predisposing cause.

Clinical Forms.—For practical purposes the cases may be divided into those with the symptoms of *acute general infection* without special localization; cases with marked *pulmonary* symptoms; and cases with *cerebral* or *cerebro-spinal* symptoms.

Other forms have been recognized, but this division covers a large majority of the cases.

Taking any series of cases it will be found that the meningeal form of acute tuberculosis exceeds in numbers the cases with general or marked pulmonary symptoms.

1. **General or Typhoid Form.**—*Symptoms.*—The patient here presents the symptoms of an infectious disease with few if any local symptoms. The cases simulate and are frequently mistaken for typhoid fever. After a period of failing health, with loss of appetite, the patient becomes feverish and weak. Occasionally the disease sets in more abruptly, but in

many instances the anamnesis closely resembles that of typhoid fever. Nose-bleeding, however, is rare. The temperature increases, the pulse becomes rapid and feeble, the tongue dry; delirium becomes marked and the cheeks are flushed. The pulmonary symptoms may be very slight; usually bronchitis exists, but not more severe than is common with typhoid fever. The pulse is seldom dicrotic, but is rapid in proportion to the pyrexia. Perhaps the most striking feature of the temperature is the irregularity; and if seen from the outset there is not the steady ascent noted in typhoid fever. There is usually an evening rise to 103°, sometimes 104°, and a morning remission of from two to three degrees. Sometimes the pyrexia is intermittent, and the thermometer may register below normal during the early morning hours. The inverse type of temperature, in which the rise takes place in the morning, is held by some writers to be more frequent in general tuberculosis than in other diseases. In rare instances there may be little or no fever. On two occasions I have had a patient admitted to my wards in a condition of profound debility, with a history of illness of from three to four weeks' duration, with rapid pulse, flushed cheeks, dry tongue, and very slight elevation in temperature, in whom (post mortem) the condition proved to be general tuberculosis. In one instance there was tolerably extensive disease at the right apex. Reinhold, from Bäumler's clinic, has recently called attention to these afebrile forms of acute tuberculosis. In nine of fifty-two cases there was no fever, or only a transient rise.

In a considerable number of these cases the respirations are increased in frequency, particularly in the early stage, and there may be signs of diffuse bronchitis and slight cyanosis. Cheyne-Stokes breathing develops toward the close.

Active delirium is rare. More commonly there are torpor and dullness, gradually deepening into coma, in which the patient dies. In some cases the pulmonary symptoms become more marked; in others, meningeal or cerebral features develop.

Diagnosis.—The differential diagnosis between general miliary tuberculosis without local manifestations and typhoid fever is extremely difficult. A point of importance, to which reference has already been made, is the irregularity of the temperature curve. The greater frequency of the respirations and the tendency to slight cyanosis is much more common in tuberculosis. There are cases, however, of typhoid fever in which the initial bronchitis is severe and may lead to dyspnœa and disturbed oxygenation. The cough may be slight or absent. Diarrhœa is rare in tuberculosis; the bowels are usually constipated; but diarrhœa may occur and persist for days. In certain cases the diagnosis has been complicated still further by the occurrence of blood in the stools. Enlargement of the spleen occurs in general tuberculosis, but is neither so early nor so marked as in typhoid fever. In children, however, the enlargement may be considerable. The urine may show traces of albumen, and unfortu-

nately Ehrlich's diazo-reaction, which is so constant in typhoid fever, is also met with in general tuberculosis. The absence of the characteristic roseola is an important feature. Occasionally in acute tuberculosis reddish spots may develop and for a time cause difficulty, but they do not come out in crops, and rarely have the characters of the true typhoid eruption. Herpes is perhaps more common in tuberculosis. Toward the close, petechiæ may appear on the skin, particularly about the wrists. A rare event is jaundice, due possibly to the eruption of tubercles in the liver.

In a few instances the presence of tubercle bacilli has been demonstrated in the blood, which in doubtful cases should therefore be examined. The spleen has been punctured and cultivations made to determine the presence or absence of the typhoid bacilli. The eye-grounds should be carefully examined for choroidal tubercles. Leucocytosis occurs in acute tuberculosis, but not in typhoid fever.

2. **Pulmonary Form.**—*Symptoms.*—From the outset the pulmonary symptoms are marked. The patient may have had a cough for months or for years without much impairment of health, or he may be known to be the subject of chronic pulmonary tuberculosis. In other instances, particularly in children, the disease follows measles and whooping-cough, and is of a distinctly broncho-pneumonic type. The disease begins with the symptoms of diffuse bronchitis. The cough is marked, the expectoration muco-purulent, occasionally rusty. Hæmoptysis has been noted in a few instances. From the outset dyspnœa is a striking feature and may be out of proportion to the intensity of the physical signs. In adults, the respirations may be as hurried as in acute pneumonia, reaching from fifty to sixty; in children, as high as eighty or more. There is more or less cyanosis of the lips and finger-tips, and the cheeks are suffused. Apart from emphysema and the later stages of severe pneumonia I know of no other pulmonary condition in which the cyanosis is so marked. The physical signs are those of bronchitis. There is rarely much alteration in the percussion note. In children there may be defective resonance at the bases, from scattered areas of broncho-pneumonia; or, what is equally suggestive, areas of hyper-resonance. Indeed, the percussion note, particularly in the front of the chest, in some cases of miliary tuberculosis, is full and clear, and it will be noted (post mortem) that the lungs are unusually voluminous. This is probably the result of more or less wide-spread acute emphysema. On auscultation, the râles are either sibilant and sonorous or small, fine, and crepitant. There may be fine crepitation from the occurrence of tubercles on the pleura (Jürgensen). In children there may be high-pitched tubular breathing at the bases or toward the root of the lung. Toward the close the râles may be larger and more mucous. The temperature rises to 102° or 103°, and may present the inverse type. The pulse is rapid and feeble. In the very acute cases the spleen is always enlarged. The disease may prove fatal in ten or twelve days, or may be protracted for weeks or even months.

Diagnosis.—The diagnosis of this form offers less difficulty and is more frequently made. There is often a history of previous cough, or the patient is known to be the subject of local disease of the lung, or of the lymph-glands, or of the bones. In children these symptoms following measles or whooping-cough indicate in the majority of cases acute miliary tuberculosis, with or without broncho-pneumonia. Occasionally the sputum contains tubercle bacilli.

The choroidal tubercle occurs in a limited number of cases and may help the diagnosis. More important in an adult is the combination of dyspnœa with cyanosis and the signs of a diffuse bronchitis. In some instances the occurrence of cerebral symptoms at once give a clew to the nature of the trouble.

3 **Meningeal Form** (*Tuberculous Meningitis*).—This affection, which is also known as acute hydrocephalus or "water on the brain," is essentially an acute tuberculosis in which the membranes of the brain, sometimes of the cord, bear the brunt of the attack.

There are several special *etiological* factors in connection with this form. It is much more common in children than in adults. It is rare during the first year of life, more frequent between the second and the fifth years. In a majority of the cases a focus of old tuberculous disease will be found, commonly in the bronchial or mesenteric glands. In a few instances the affection seems to be primary in the meninges. It is very difficult, however, in an ordinary post-mortem to make an exhaustive search, and the lesion may be in the bones, sometimes in the middle ear, or in the genito-urinary organs. In those instances in which no primary focus has been discovered it has been suggested that the bacilli reach the meninges through the cribriform plate of the ethmoid from the upper part of the nostrils, but this is not probable.

Morbid Anatomy.—Tuberculous meningitis presents a very characteristic picture. The meninges at the base are most involved, hence the term basilar meningitis. The parts about the optic chiasm, the Sylvian fissures, and the interpeduncular space are affected. There may be only slight turbidity and matting of the membranes, and a certain stickiness with serous infiltration; but more commonly there is a turbid exudate, fibrino-purulent in character, which covers the structures at the base, surrounds the nerves, extends out in the Sylvian fissures, and appears on the lateral, rarely on the upper, surfaces of the hemispheres. The tubercles may be very apparent, particularly in the Sylvian fissures, appearing as small, whitish nodules on the membranes. They vary much in number and size, and may be difficult to find. The amount of exudate bears no definite relation to the abundance of tubercles. The arteries of the anterior and posterior perforated spaces should be carefully withdrawn and searched, as upon them nodular tubercles may be found when not present elsewhere. In doubtful cases the middle cerebral arteries should be very carefully removed, spread on a glass plate with a black background, and examined

with a low objective. The tubercles are then seen as nodular enlargements on the smaller arteries. The lateral ventricles are dilated (acute hydrocephalus) and contain a turbid fluid; the ependyma may be softened, and the septum lucidum and fornix are usually broken down. The convolutions are often flattened and the sulci obliterated owing to the increased intra-ventricular pressure. Histologically the tubercles are seen to develop in the perivascular sheaths, producing circumscribed aggregations of lymphoid and epithelioid cells. The lumen of the vessel is narrowed and thrombosis may result. The meninges are not alone involved, but the contiguous cerebral substance is more or less œdematous and infiltrated with leucocytes, so that anatomically the condition is in reality a *meningo-encephalitis*.

There are instances in which the acute process is associated with chronic meningeal tuberculosis; cases which may for months present the clinical picture of brain tumor.

Although in a majority of instances the process is cerebral, the spinal meninges may also be involved, particularly those of the cervical cord. There are cases indeed in which the symptoms are chiefly spinal. A sailor, who had fallen on the deck three weeks before his death, was admitted to the Montreal General Hospital. He presented signs of meningitis, chiefly spinal, which were naturally attributed to traumatism. The post-mortem showed absence of tubercles and lymph at the base of the brain, and an extensive eruption of miliary tubercles with much turbid lymph over the entire spinal meninges. There were small cheesy masses at the apices of the lungs.

Symptoms.—Tuberculous meningitis presents an extremely complex clinical picture. It will be best to describe the form found in children.

Prodromal symptoms are common. The child may have been in failing health for some weeks, or may be convalescent from measles or whooping-cough. In many instances there is a history of a fall. The child gets thin, is restless, peevish, irritable, loses its appetite, and the disposition may completely change. Symptoms pointing to the disease may then set in, either quite suddenly with a convulsion, or more commonly with headache, vomiting, and fever, three essential symptoms of the onset which are rarely absent. The pain may be intense and agonizing. The child puts its hand to its head and occasionally, when the pain becomes worse, gives a short, sudden cry, the so-called hydrocephalic cry. Sometimes the child screams continuously until utterly exhausted. I saw in West Philadelphia a case of basilar meningitis in a girl of thirteen, who for three days, when not under the influence of a powerful sedative or of chloroform, screamed at the top of her voice so as to be heard a square or more away. The vomiting is without apparent cause, and is independent of taking of food. Constipation is usually present. The fever is slight, but gradually rises to 102° or 103°. The pulse is at first rapid, subsequently irregular and slow. The respirations are rarely altered. During

sleep the child is restless and disturbed. There may be twitchings of the muscles, or sudden startings; or the child may wake up from sleep in great terror. In this early stage the pupils are usually contracted. These are the chief symptoms of the initial stage, or, as it is termed, the *stage of irritation.*

In the second period of the disease these irritative symptoms subside; vomiting is no longer marked, the abdomen becomes retracted, boat-shaped or *carinated.* The bowels are obstinately constipated, the child no longer complains of headache, but is dull and apathetic, and when roused is more or less delirious. The head is often retracted and the child utters an occasional cry. The pupils are dilated or irregular, and a squint may develop. Sighing respiration is common. Convulsions may occur, or rigidity of the muscles of one side or of one limb. The temperature is variable, ranging from 100° to 102·5°. A blotchy erythema is not uncommon on the skin. If the finger-nail is drawn across the skin of any region a red line comes out quickly, the so-called *tâche cérébrale*, which, however, has no diagnostic significance.

In the final period, or stage of *paralysis*, the coma increases and the child cannot be roused. Convulsions are not infrequent, and there are spasmodic contractions of the muscles of the back and neck. Spasms may occur in the limbs of one side. Optic neuritis and paralysis of the ocular muscles may be present. The pupils become dilated, the eyelids are only partially closed, and the eyeballs are rolled up so that the corneæ are only covered in part by the upper eyelid. Diarrhœa may develop, the pulse becomes rapid, and the child may sink into a typhoid state with dry tongue, low delirium, and involuntary passages of urine and fæces. The temperature often becomes subnormal, sinking in rare instances to 93° or 94°. In some cases there is ante-mortem elevation of temperature, the fever rising to 106°. The entire duration of the disease is from a fortnight to three or four weeks.

There are cases of tuberculous meningitis which pursue a more rapid course. They set in with great violence, often in persons apparently in good health, and may prove fatal within a few days. In these instances, more commonly seen in adults, the convex surface of the brain is usually involved. There are again instances which are essentially chronic and display symptoms of a limited meningitis; sometimes with pronounced psychical symptoms, and sometimes with those of cerebral tumor.

There are certain features which call for special comment.

The irregularity and slowness of the pulse in the early and middle stages of the disease are points upon which all authors agree. Toward the close, as the heart's action becomes weaker, the pulsations are more frequent. The temperature is usually elevated, but there are instances in which it does not rise in the whole course of the disease much above 100°. It may be extremely irregular, and the oscillations are often as much as three or four degrees in the day. Toward the close the temperature may

sink to 95°, occasionally to 94°, or there may be hyperpyrexia. In a case of Bäumler's the temperature rose before death to 43·7° C. (110·7° F.).

The ocular symptoms of the disease are of special importance. In the early stages narrowing of the pupils is the rule. Toward the close, with increase in the intra-cranial pressure, the pupils dilate and are irregular. There may be conjugate deviation of the eyes. Of ocular palsies the third nerve is most frequently involved. The changes in the eye-grounds are very important. Neuritis is the most common. According to Gowers, the disk at first becomes full colored and has hazy outlines, and the veins are dilated. Swelling and striation become pronounced, but the neuritis is rarely intense. Of twenty-six cases studied by Garlick, in six the condition was of diagnostic value. The tubercles in the choroid are rare and much less frequently seen during life than post-mortem figures would indicate. Thus Litten found them (post mortem) in thirty-nine out of fifty-two cases. They were present in only one of the twenty-six cases of tuberculous meningitis examined by Garlick. I have never met them clinically, and have only found two instances post mortem. Heinzel examined with negative results forty-one cases.

Among the motor symptoms convulsions are most common, but there are other changes which deserve special mention. A tetanic contraction of one limb may persist for several days, or a cataleptic condition. Tremor and athetoid movements are sometimes seen. The paralyses are either hemiplegias or monoplegias. Hemiplegia may result from disturbance in the cortical branches of the middle cerebral artery, occasionally from softening in the internal capsule, due to involvement of the central branches. Of monoplegias, that of the face is perhaps most common, and if on the right side it may occur with aphasia. In two of my cases in adults aphasia developed. Brachial monoplegia may be associated with it. In the more chronic cases the symptoms persist for months, and there may be a characteristic Jacksonian epilepsy when the tubercles involve the meninges of the motor cortex.

The *prognosis* in this form of meningitis is always most serious. I have neither seen a case which I regarded as tuberculous recover, nor have I seen post-mortem evidence of past disease of this nature. Cases of recovery have been reported by reliable authorities, but they are extremely rare, and there is always a reasonable doubt as to the correctness of the diagnosis. The differential features will be considered in connection with acute meningitis.

III. Tuberculosis of the Lymph-glands (*Scrofula*).

Scrofula is tubercle, as it has been shown that the bacillus of Koch is the essential element. It is not yet definitely settled whether the virus which produces the chronic adenitis or scrofula differs from that which produces tuberculosis in other parts, or whether it is the local conditions

in the glands which account for the slow development and milder course. The experiments of Arloing would indicate that the virus was attenuated or milder, for he has shown that the caseous material of a lymph-gland killed guinea-pigs, while rabbits escaped. The guinea-pig, as is well known, is the more susceptible animal of the two. The observations of Lingard are still more conclusive, as showing a variation in the virulence of the tubercle bacillus. Guinea-pigs inoculated with ordinary tubercle showed lymphatic infection within the first week, and the animals died within three months; infected with material from scrofulous glands, the lymphatic enlargement did not appear until the second or third week, and the animals survived for six or seven months. He showed, moreover, that the virulence of the infection obtained from the scrofulous glands increased in intensity by passing through a series of guinea-pigs. Eve's experiments show that scrofulous material invariably produces tuberculosis in guinea-pigs and very often in rabbits.

Tuberculous adenitis is met with at all ages. It is more common in children than in adults, but it is not infrequent in the middle period of life, and may occur in old age.

The tubercle bacillus is ubiquitous. All are exposed to infection, and upon the local conditions, whether favorable or unfavorable, depend the fate of those organisms which find lodgment in our bodies. It is possible, of course, that tuberculous adenitis may be congenital, but such instances must be extremely rare. A special predisposing factor in lymphatic tuberculosis is catarrhal inflammation of the mucous membranes, which in itself excites slight adenitis of the neighboring glands. In a child with constantly recurring naso-pharyngeal catarrh, the bacilli which lodge on the mucous membranes find in all probability the gateways less strictly guarded and are taken up by the lymphatics and passed to the nearest glands. In conditions of health the local resistance, or, as some would put it, the phagocytes, would be active enough to deal with the invaders, but the irritation of a chronic catarrh weakens the resistance of the lymph-tissue and the bacilli are enabled to develop and gradually to change a simple into a tuberculous adenitis. The frequent association of tuberculous adenitis of the bronchial glands with whooping-cough and with measles, and the frequent development of tubercle in the mesenteric glands in children with intestinal catarrh, find in this way a rational explanation. After all, as Virchow pointed out, an increased vulnerability of the tissue, however brought about, is the important factor in the disease.

The following are some of the features of interest in tuberculous adenitis:

(*a*) The local character of the disease; thus, the glands of the neck, or at the bifurcation of the bronchi, or those of the mesentery, may be alone involved.

(*b*) The tendency to spontaneous healing. In a large proportion of the cases the battle which ensues between the bacilli and the tissue-cells is

long; but the latter are finally successful, and we find in the calcified remnants in the bronchial and mesenteric lymph-glands evidences of victory. Too often in the bronchial glands a truce only is declared and hostilities may break out afresh in the form of an acute tuberculosis.

(*c*) The tendency of tuberculous adenitis to pass on to suppuration. The frequency with which, particularly in the glands of the neck, we find the tuberculous processes associated with pus is a special feature of this form of adenitis. In nearly all instances the pus is sterile. Whether the suppuration is excited by the bacilli or by their products, or whether it is the result of a mixed infection with pus organisms, which are subsequently destroyed, has not been settled.

(*d*) The existence of an unhealed focus of tuberculous adenitis is a constant menace to the organism. It is safe to say that in three fourths of the instances of acute tuberculosis the infection is derived from this source. On the other hand, it has been urged that scrofula in childhood gives a sort of protection against tuberculosis in adult life. We certainly do meet with many persons of exceptional bodily vigor who in childhood had enlarged glands, but the evidence which Marfan * brings forward in support of this view is not conclusive.

Clinical Forms.—1. **General Tuberculous Lymphadenitis.**—In exceptional instances we find diffuse tuberculosis of nearly all the lymph-glands of the body with little or no involvement of other parts. The most extreme cases of it which I have seen have been in negro patients. Two well-marked cases occurred at the Philadelphia Hospital. In one, a woman, aged thirty-four, was admitted April 4th, with enlarged glands in the right side of the neck and irregular fever. The chart from April, 1888, until March, 1889, showed persistent fever, ranging from 101° to 103°, occasionally rising to 104°. On December 16th the glands on the right side of the neck were removed. After an attack of erysipelas, on February 17th, she gradually sank and died March 5th. The lungs presented only one or two puckered spots at the apices. The bronchial, retro-peritoneal, and mesenteric glands were greatly enlarged and caseous. No intestinal, uterine, or bone disease. The continuous high fever in this case depended apparently upon the tuberculous adenitis, which was much more extensive than was supposed during life. In these instances the enlargement is most marked in the retro-peritoneal, bronchial, and mesenteric glands, but may be also present in the groups of external glands. Occurring acutely, it presents a picture resembling Hodgkin's disease. In a case which died in the Montreal General Hospital this diagnosis was made. The cervical and axillary glands were enormously enlarged, and death was caused by infiltration of the larynx.

2. **Local Tuberculous Adenitis.**—(*a*) *Cervical.*—This is the most common form met with in children. It is seen particularly among the poor

* Archives générales, 1886.

and those who live continuously in the impure atmosphere of badly ventilated lodgings. Children in foundling hospitals and asylums are specially prone to the disease. In this country it is most common in the negro race. As already stated, it is often met with in catarrh of the nose and throat, or chronic enlargement of the tonsils; or the child may have had eczema of the scalp or a purulent otitis.

The submaxillary glands are first involved, and are popularly spoken of as enlarged *kernels*. They are usually larger on one side than on the other. As they increase in size, the individual tumors can be felt; the surface is smooth and the consistence firm. They may remain isolated, but more commonly they form large, knotted masses, over which the skin is, as a rule, freely movable. In many cases the skin ultimately becomes adherent, and inflammation and suppuration occur. An abscess points and, unless opened, bursts, leaving a sinus which heals slowly. The disease is frequently associated with coryza, with eczema of the scalp, ear, or lips, and with conjunctivitis or keratitis. When the glands are large and growing actively, there is fever. The subjects are usually anæmic, particularly if suppuration has occurred. The progress of this form of adenitis is slow and tedious. Death, however, rarely follows, and many aggravated cases in children ultimately get well. Not only the submaxillary group, but the glands above the clavicle and in the posterior cervical triangle, may be involved. In other instances the cervical and axillary glands are involved together, forming a continuous chain which extends beneath the clavicle and the pectoral muscle. With them the bronchial glands may also be enlarged and caseous. Not infrequently the enlargement of the supraclavicular and axillary group of glands on one side precedes the development of a tuberculous pleurisy or of pulmonary tuberculosis.

(*b*) *Bronchial.*—The mediastinal lymph-glands constitute filters in which lodge the various foreign particles which escape the normal phagocytes of bronchi and lungs. Among these foreign particles, and probably attached to them, tubercle bacilli are not uncommon, and we find tubercles and caseous matter with great frequency in the mediastinal glands, particularly those about the bronchi. It is stated that this process is always secondary to a focus, however small, in the lungs, but my experience does not bear out such a statement. As already mentioned, Northrup found them involved in every one of a hundred and twenty-seven cases at the New York Foundling Hospital. This tuberculous adenitis may, in the bronchial glands, attain the dimensions of a tumor of large size. But even when this occurs there may be no pressure symptoms. In children the bronchial adenitis is apt to be associated with suppuration.

A more serious danger in tuberculous disease of the bronchial glands is systemic infection, which takes place through the vessels. Local infection of the lungs may also occur. In the tuberculous broncho-pneumonia of children it is usual to find the bronchial glands enormously en-

larged, passing deeply into the hilus, adjoining, and in some instances even merging with, areas of caseation of the pulmonary tissue itself.

There is a special danger of infection of the pericardium by tuberculous lymph-glands in the anterior mediastinum.

(*c*) *Mesenteric; Tabes mesenterica.*—In this affection, the abdominal scrofula of old writers, the glands of the mesentery and retro-peritonæum become enlarged and caseate; more rarely they suppurate or calcify. A slight tuberculous adenitis is extr mely common in children, and is often accidentally found (post mortem) when the children have died of other diseases. It may be a primary lesion associated with intestinal catarrh, or it may be secondary to tuberculous disease of the intestines.

The primary cases are very common in children, as may be gathered from Woodhead's figures. The general involvement of the glands interferes seriously with nutrition, and the patients are puny, wasted, and anæmic. The abdomen is enlarged and tympanitic; diarrhœa is a constant feature; the stools are thin and offensive. There is moderate fever, but the general wasting and debility are the most characteristic features. The enlarged glands cannot often be felt, owing to the distended condition of the bowels. These cases are often spoken of as consumption of the bowels, but in a majority of them the intestines do not present tuberculous lesions. In a considerable number of the cases of tabes mesenterica the peritonæum is also involved, and in such the abdomen is large and hard, and nodules may be felt. The condition is one to which the French have given the name *carreau.*

In adults tuberculous disease of the mesenteric glands may occur as a primary affection, or in association with pulmonary disease. Gairdner* gives a remarkable instance of the kind in a man aged twenty-one. Instances of this sort are not uncommon in the literature. Large tumors may exist without tuberculous disease in the intestines or in any other parts.

The diagnosis of local and general tuberculous adenitis from lymphadenoma will be subsequently considered.

IV. Pulmonary Tuberculosis (*Phthisis, Consumption*).

Three clinical groups may be conveniently recognized: (1) *tuberculo-pneumonic phthisis*—acute phthisis; (2) *chronic ulcerative phthisis;* and (3) *fibroid phthisis.*

According to the mode of infection there are two distinct types of lesions:

(*a*) When the bacilli reach the lungs through the blood-vessels, the primary lesion is usually in the tissues of the alveolar walls, in the capillary vessels, the epithelium of the air-cells, and in the connective-tissue

* Lectures to Practitioners, Gairdner and Coats, 1888.

framework of the septa. The process of cell division proceeds as already described in the general histology of tubercle. The irritation of the bacilli produces, within a few days, the small, gray miliary nodules, involving several alveoli and consisting largely of round, cuboidal, uninuclear epithelioid cells. Depending upon the number of bacilli which reach the lung in this way, either a localized or a general tuberculosis is excited. The tubercles may be uniformly scattered through both lungs and form a part of a general miliary tuberculosis, or they may be confined to the lungs, or even in great part to one lung. The changes which the tubercles undergo have already been referred to. The further changes may be: (1) Arrest of the process of cell division, gradual sclerosis of the tubercle, and ultimately complete fibroid transformation. (2) Caseation of the centre of the tubercle, extension at the periphery by proliferation of the epithelioid and lymphoid cells, so that the individual tubercles or small groups become confluent and form diffuse areas which undergo caseation and softening. (3) Occasionally as a result of intense infection of a localized region through the blood-vessels the tubercles are thickly set. The intervening tissue becomes acutely inflamed, the air-cells are filled with the products of a desquamative pneumonia, and many lobules are involved.

(*b*) When the bacilli reach the lung through the bronchi—inhalation tuberculosis—the picture differs. The smaller bronchi and bronchioles are more extensively affected; the process is not confined to single groups of alveoli, but has a more lobular arrangement, and the tuberculous masses from the outset are larger, more diffuse, and may in some cases involve an entire lobe or the greater part of a lung. It is in this mode of infection that we see the characteristic peri-bronchial granulations and the areas of the so-called nodular broncho-pneumonia. These broncho-pneumonic areas, with on the one hand caseation, ulceration, and cavity formation, and on the other sclerosis and limitation, make up the essential elements in the anatomical picture of tuberculous phthisis.

1. Acute Pneumonic Phthisis.

This form, known also by the name of galloping consumption, is met with both in children and adults. In the former many of the cases are mistaken for simple broncho-pneumonia.

Two types may be recognized, the *pneumonic* and *broncho-pneumonic*.

(*a*) In the *pneumonic form* one lobe may be involved, or in some instances an entire lung. The organ is heavy, the affected portion airless, the pleura usually covered with thin exudation, and on section the picture resembles closely that of ordinary hepatization. The following is an extract from the post-mortem report of a case in which death occurred twenty-nine days after the onset of the illness, having all the characters of an acute pneumonia: "Left lung weighs 1,500 grammes (double the weight of the

other organ) and is heavy and airless, crepitant only at the anterior margins. Section shows a small cavity the size of a walnut at the apex, about which are scattered tubercles in a consolidated tissue. The greater part of the lung presents a grayish-white appearance due to the aggregation of tubercles which in some places have a continuous, uniform appearance, in others are surrounded by an injected and consolidated lung-tissue. Toward the margins of the lower lobe strands of this firm reddish tissue separate anæmic, dry areas. There are in the right lung three or four small groups of tubercles but no caseous masses. The bronchial glands are not tuberculous." Here the intense local infection was due to the small focus at the apex of the lung, probably an aspiration process.

Only the most careful inspection may reveal the presence of miliary tubercles, or the attention may be arrested by the detection of tubercles in the other lung or in the bronchial glands. The process may involve only one lobe. There may be older areas which are of a peculiarly yellowish-white color and distinctly caseous. The most remarkable picture is presented by cases of this kind in which the disease lasts for some months. A lobe or an entire lung may be enlarged, firm, airless throughout, and converted into a dry, yellowish-white, cheesy substance. Cases are met with in which the entire lung from apex to base is in this condition, with perhaps only a small, narrow area of air-containing tissue on the margin. More commonly, if the case has lasted for two or three months, rapid softening has taken place at the apex. The following brief extract gives the actual condition of the lung in a case in which death occurred in the eleventh week: "Left lung is solid and heavy, weighing 1,490 grammes, and is nowhere crepitant. The upper third of the upper lobe is occupied by a cavity, containing blood and pus, the walls of which are formed by ragged caseous masses. The rest of the lung is firm and solid, and on section presents a uniform opaque white color. The surface is dry, and all parts present the same cheesy appearance."

Symptoms.—The attack sets in abruptly with a chill, usually in an individual who has enjoyed good health, although in many cases the onset has been preceded by exposure to cold, or there have been debilitating circumstances. The temperature rises rapidly after the chill, there are pain in the side, and cough, with at first mucoid, subsequently rusty-colored expectoration. The dyspnœa may become extreme and the patient may have suffocative attacks. The physical examination shows involvement of one lobe or of one lung, with signs of consolidation, dulness, increased fremitus, at first feeble or suppressed vesicular murmur, and subsequently well-marked bronchial breathing. The upper or lower lobe may be involved, or in some cases the entire lung.

At this time, as a rule, no suspicion enters the mind of the practitioner that the case is anything but one of frank lobar pneumonia. Occasionally there may be suspicious circumstances in the history of the patient or in his family; but, as a rule, no stress is laid upon them in comparison

with the intense and characteristic mode of onset. Between the eighth and tenth day, instead of the expected crisis, the condition becomes aggravated, the temperature is irregular, and the pulse more rapid. There may be sweating, and the expectoration becomes muco-purulent. Even in the second or third week, with the persistence of these symptoms, the physician tries to console himself with the idea that the case is one of unresolved pneumonia, and that all will yet be well. Gradually, however, the severity of the symptoms, the presence of physical signs indicating softening, the existence of elastic tissue and tubercle bacilli in the sputa present the mournful proofs that the case is one of acute pneumonic phthisis. Death may occur before softening takes place, even in the second or third week. In other cases there is extensive destruction at the apex, with rapid formation of cavity, and the case may drag on for two or three months.

Diagnosis.—It is by no means widely recognized in the profession that there is a form of acute phthisis which may closely simulate ordinary pneumonia. Waters, of Liverpool, gave an admirable description of these cases and called attention to the difficulty in distinguishing them from ordinary pneumonia. Certainly the mode of onset affords no criterion whatever. A healthy, robust-looking young Irishman, a cab-driver, who had been kept waiting on a cold, blustering night until three in the morning, was seized the next afternoon with a violent chill, and the following day was admitted to my wards at the University Hospital, Philadelphia. He was made the subject of a clinical lecture on the fifth day, when there was absent no single feature in history, symptoms, or physical signs of acute lobar pneumonia of the right upper lobe. It was not until ten days later, when bacilli were found in his expectoration, that we were made aware of the true nature of the case. I know of no criterion by which cases of this kind can be distinguished in the early stage. The tubercle bacilli are not present at first. A point to which Traube called attention, and which is also referred to as important by Herard and Cornil, is the absence of bronchial sounds in the consolidated region; but this, I am sure, does not hold good in all cases. The tubular breathing may be intense and marked as early as the fourth day; and again, how common it is to have, as one of the earliest and most suggestive symptoms of lobar pneumonia, suppression or enfeeblement of the vesicular murmur! In many cases, however, there are suspicious circumstances in the onset; the patient has been in bad health, or may have had previous pulmonary trouble, or there are recurring chills. Careful examination of the sputa and a study of the physical signs from day to day can alone determine the true nature of the case. A point of some moment is the character of the fever, which in true pneumonia is more continuous, particularly in severe cases, whereas in this form of tuberculosis remissions of 1·5° or 2° are not infrequent.

(*b*) *Acute tuberculous broncho-pneumonia* is more common, particu-

larly in children, and forms a majority of the cases of *phthisis florida* or "galloping consumption." It is an acute caseous broncho-pneumonia, starting in the smaller tubes, which become blocked with a cheesy substance, while the air-cells of the lobule are filled with the products of a catarrhal pneumonia. In the early stage the areas have a grayish-red, later an opaque-white, caseous appearance. By the fusion of contiguous masses an entire lobe may be rendered nearly solid, but there can usually be seen between the groups areas of crepitant air tissue. This is not an uncommon picture in the acute phthisis of adults, but it is still more frequent in children. The following is an extract from the post-mortem of a case on a child aged four months, which died in the sixth week of illness: "The upper lobe of the right lung is scarcely anywhere crepitant except at the anterior edge. The middle and lower lobes are heavy and slightly crepitant; the visceral pleura is beset with tubercles which have grown into it from the lung. On section the right upper lobe is occupied with caseous masses from five to twelve millimetres in diameter, separated from each other by an intervening tissue of deep-red color. The bronchi are filled with cheesy substance. The middle and lower lobes are stuffed with tubercles, many of which are becoming caseous. Toward the diaphragmatic surface of the lower lobe there is a small cavity, the size of a marble. The left lung is more crepitant and uniformly studded with tubercles of all sizes, some as large as peas. There is an acute tuberculous bronchitis in the smaller and larger branches, and extending into the trachea. The bronchial glands are very large, and one contains a tuberculous abscess."

In children the enlarged bronchial glands usually surround the root of the lung, and even pass deeply into the substance, and the lobules are often involved by direct contact.*

In other cases the caseous broncho-pneumonia involves groups of alveoli or lobules in different portions of the lungs, more commonly at both apices, forming areas from one to three centimetres in diameter. The size of the mass depends largely upon that of the bronchus involved. There are cases which probably should come in this category, in which, with a history of an acute illness of from four to eight weeks, the lungs are extensively studded with large gray tubercles, ranging in size from five to ten millimetres. In some instances there are cheesy masses the size of a cherry. All of these are grayish-white in color, distinctly cheesy, and between the adjacent ones, particularly in the lower lobe, there may be recent pneumonia, or the condition of lung which has been termed splenization. In a case of this kind at the Philadelphia Hospital death took place about the eighth week from the abrupt onset of the illness with hæmorrhage. There were no extensive areas of consolidation, but the cheesy nodules were uniformly scattered throughout both lungs. No softening had taken place.

* *Vide* the drawings illustrating Northrup's article; New York Medical Journal, February 21, 1891.

Symptoms.—The symptoms of acute broncho-pneumonic phthisis are very variable. In adults the disease may attack persons in good health, but who are overworked or "run down" from any cause. Hæmorrhage initiates the attack in a few cases. There may be repeated chills; the temperature is high, the pulse rapid, and the respirations are increased. The loss of flesh and strength is very striking.

The physical signs may at first be uncertain and indefinite, but finally there are areas of impaired resonance, usually at the apices; the breath-sounds are harsh and tubular, with numerous râles. The sputa may early show elastic tissue and tubercle bacilli. In the acute cases, within three weeks, the patient may be in a marked typhoid state, with delirium, dry tongue, and high fever. Death may occur within three weeks. In other cases the onset is severe, with high fever, rapid loss of flesh and strength, and signs of extensive unilateral or bilateral disease. Softening takes place; there are sweats, chills, and progressive emaciation, and all the features of *phthisis florida*. Six or eight weeks or later the patient may begin to improve, the fever lessens, the general symptoms mitigate, and a case which looked as if it would certainly terminate fatally within a few weeks drags on and becomes chronic.

In *children* the disease most commonly follows the infectious diseases, particularly measles and whooping-cough.* The profession is gradually recognizing the fact that a majority of all such cases are tuberculous. At least *three groups* of these cases of tuberculous broncho-pneumonia may be recognized. In the *first* the child is taken ill suddenly while teething or during convalescence from fever; the temperature rises rapidly, the cough is severe, and there may be signs of consolidation at one or both apices with râles. Death may occur within a few days, and the lung shows areas of broncho-pneumonia, with perhaps here and there scattered opaque grayish-yellow nodules. Macroscopically the affection does not look tuberculous, but histologically miliary granulations and bacilli may be found.† Tubercles are usually present in the bronchial glands, but the appearance of the broncho-pneumonia may be exceedingly deceptive, and it may require careful microscopical examination to determine its tuberculous character. The *second group* is represented by the case of the child previously quoted, which died at the sixth week with the ordinary symptoms of severe broncho-pneumonia. And the *third group* is that in which, during the convalescence from an infectious disease, the child is taken ill with fever, cough, and shortness of breath. The severity of the symptoms mitigates within the first fortnight; but there is loss of flesh, the general condition is bad, and the physical examination shows the presence of scattered râles throughout the lungs, and here and there areas of defective resonance. The child has sweats, the fever becomes hectic in

* "Tussis convulsiva vestibulum tabis" (Willis).

† Cornil and Babes, Les Bactéries, tome ii, 1890.

character, and in many cases the clinical picture gradually develops into that of chronic phthisis.

2. Chronic Ulcerative Phthisis.

Under this heading may be grouped the great majority of cases of pulmonary tuberculosis, in which the lesions proceed to ulceration and softening, and ultimately produce the well-known picture of chronic phthisis. At first a strictly tuberculous affection, it ultimately becomes, in a majority of cases, a mixed disease, many of the most prominent symptoms of which are due to septic infection from purulent foci and cavities.

Morbid Anatomy.—Inspection of the lungs in a case of chronic phthisis shows a remarkable variety of lesions, comprising nodular tubercles, diffuse tuberculous infiltration, caseous masses, pneumonic areas, cavities of various size, with changes in the pleura, bronchi, and bronchial glands.

1. **The Distribution of the Lesions.**—For years it has been recognized that the most advanced lesions are at the apices, and that the disease progresses downward, usually more rapidly in one of the lungs. This general statement, which has passed current in the text-books ever since the masterly description of Laennec, has recently been carefully elaborated by Kingston Fowler, who finds that the disease in its onward progress through the lungs follows, in a majority of the cases, distinct routes. In the upper lobe the primary lesion is not, as a rule, at the extreme apex, but from an inch to an inch and a half below the summit of the lung, and nearer to the posterior and external borders. The lesion here tends to spread downward, probably from inhalation of the virus, and this accounts for the frequent circumstance that examination behind, in the supraspinus fossa, will give indications of disease before any evidences exist at the apex in front. Anteriorly this initial focus corresponds to a spot just below the centre of the clavicle, and the direction of extension in front is along the anterior aspect of the upper lobe, along a line running about an inch and a half from the inner ends of the first, second, and third interspaces. A second less common site of the primary lesion in the apex "corresponds on the chest wall with the first and second interspaces below the outer third of the clavicle." The extension is downward, so that the outer part of the upper lobe is chiefly involved.

In the middle lobe of the right lung the affection usually follows the upper lobe on the same side. In the involvement of the lower lobe the first secondary infiltration is about an inch to an inch and a half below the posterior extremity of its apex, and corresponds on the chest wall to a spot opposite the fifth dorsal spine. This involvement is of the greatest importance clinically, as "in the great majority of cases, when the physical signs of the disease at the apex are sufficiently definite to allow of the diagnosis of phthisis being made, the lower lobe is already affected." Ex-

amination, therefore, should be made carefully of this posterior apex in all suspicious cases. In this situation the lesion spreads downward and laterally along the line of the interlobular septa, a line which is marked by the vertebral border of the scapula, when the hand is placed on the opposite scapula and the elbow raised above the level of the shoulder. Once present in an apex, the disease usually extends in time to the opposite upper lobe; but not, as a rule, until the apex of the lower lobe of the lung first affected has been attacked.

Of 427 cases above mentioned, the right apex was involved in 172, the left in 130, both in 111.

Lesions of the base may be primary, though this is rare. Percy Kidd makes the proportion of basic to apicic phthisis one to five hundred, a smaller number than existed in my series. In very chronic cases there may be arrested lesions at the apex and more recent lesions at the base.

2. **Summary of the Lesions in Chronic Ulcerative Phthisis.**—(*a*) *Miliary Tubercles.*—These may not be evident on microscopical examination, or there may be a few colonies, "the secondary crop" of Laennec, about the caseous areas. In other instances, with old lesions at the apex, there are, throughout the lower lobes, scattered groups of miliary tubercles which have undergone fibroid and pigmentary changes. Sometimes, in cases with cavity formation at the apex, the greater part of the lower lobes present many groups of firm, sclerotic, miliary tubercles, which may indeed form the distinguishing anatomical feature—a chronic miliary tuberculosis.

(*b*) *Tuberculous Broncho-pneumonia.*—In a large proportion of the cases of chronic phthisis the terminal bronchiole is the point of origin of the process, consequently we find the smaller bronchi and their alveolar territories blocked with the accumulated products of inflammation in all stages of *caseation.* At an early period a cross-section of an area of tuberculous broncho-pneumonia gives the most characteristic appearance. The central bronchiole is seen as a small orifice, or it is plugged with cheesy contents, while surrounding it is a caseous nodule, the so-called peribronchial tubercle. The longitudinal section has a somewhat dendritic or foliaceous appearance. The condition of the picture depends much upon the slowness or rapidity with which the process has advanced. The following changes may occur:

Ulceration.—When the caseation takes place rapidly or ulceration occurs in the bronchial wall, the mass may break down and form a small cavity.

Sclerosis.—In other instances the process is more chronic. Fibroid changes gradually produce a sclerosis of the affected area, a condition which is sometimes called *cirrhosis nodosa tuberculosa.* The sclerosis may be confined to the margin of the mass, forming a limiting capsule, within which is a uniform, firm, cheesy substance, in which lime salts are often deposited. This represents the healing of one of these areas of caseous

broncho-pneumonia. It is only, however, when complete fibroid transformation or calcification has occurred that we can really speak of healing. In many instances the colonies of miliary tubercles about these masses show that the virus is still active in them. Subsequently, in ulcerative processes, these calcareous bodies — lung-stones, as they are sometimes called—may be expectorated.

(*c*) *Pneumonia.*—An important though secondary place is occupied by inflammation of the alveoli surrounding the tubercles, which become filled with epithelioid cells. The consolidation may extend for some distance about the tuberculous foci and unite them into areas of uniform consolidation. Although in some instances this inflammatory process may be simple, in others it is undoubtedly specific. It is excited by the tubercle bacilli and is a manifestation of their action. It may present a very varied appearance; in some instances resembling closely ordinary red hepatization, in others more homogeneous and infiltrated, the so-called *infiltration tuberculeuse* of Laennec. In other cases the contents of the alveoli undergo fatty degeneration, and appear on the cut surface as opaque white or yellowish-white bodies. In early phthisis much of the consolidation is due to this pneumonic infiltration, which may surround for some distance the smaller tuberculous foci.

(*d*) *Cavities.*—A vomica is a cavity in the lung tissue, produced by necrosis and ulceration. It differs materially from the bronchiectatic form. The process usually begins in the wall of the bronchus in a tuberculous area. Dilatation is produced by retained secretion, and necrosis and ulceration of the wall occur with gradual destruction of the contiguous tissues. By extension of the necrosis and ulceration the cavity increases, contiguous ones unite, and in an affected region there may be a series of small excavations communicating with a bronchus. In nearly all instances the process extends from the bronchi, though it is possible for necrosis and softening to take place in the centre of a caseous area without primary involvement of the bronchial wall. Three forms of cavities may be recognized:

The *fresh ulcerative*, seen in acute phthisis, in which there is no limiting membrane, but the walls are made up of softened, necrotic, and caseous masses. Small vomicæ of this sort, situated just beneath the pleura, may rupture and cause pneumothorax. In cases of acute tuberculo-pneumonic phthisis they may be large, occupying the greater portion of the upper lobe. In the chronic ulcerative phthisis, cavities of this sort are invariably present in those portions of the lung in which the disease is advancing. At the apex there may be a large old cavity with well-defined walls, while at the anterior margin of the upper lobes, or in the apices of the lower lobe, there are recent ulcerating cavities communicating with the bronchi.

Cavities with well-defined walls.—A majority of the cavities in the chronic form of phthisis have a well-defined limiting membrane, the

inner surface of which constantly produces pus. The walls are crossed by trabeculæ which represent remnants of bronchi and blood-vessels. Even the vomicæ with the well-defined walls extend gradually by a slow necrosis and destruction of the contiguous lung tissue. The contents are usually purulent, similar in character to the grayish nummular sputa coughed up by phthisical patients. Not infrequently the membrane is vascular or it may be hæmorrhagic. Occasionally, when gangrene has occurred in the wall, the contents are horribly fœtid. These cavities may occupy the greater portion of the apex, forming an irregular series which communicate with each other and with the bronchi, or the entire upper lobe except the anterior margin may be excavated, forming a thin-walled cavity. In rare instances the process has proceeded to total excavation of the lung, not a remnant of which remains, except perhaps a narrow strip at the anterior margin. In a case of this kind, in a young girl, the cavity held forty fluidounces.

Quiescent Cavities.—When quite small and surrounded by dense cicatricial tissue communicating with the bronchi they form the *cicatrices fistuleuses* of Laennec. Occasionally one apex may be represented by a series of these small cavities, surrounded by dense fibrous tissue. The lining membrane of these old cavities may be quite smooth, almost like a mucous membrane. Cavities of any size do not heal completely.

Cases are often seen in which it has been supposed that a cavity has healed; but the signs of excavation are notoriously uncertain, and there may be pectoriloquy and cavernous sounds with gurgling, resonant râles in an area of consolidation close to a large bronchus.

In the formation of vomicæ the blood-vessels gradually become closed by an obliterating inflammation. They are the last structures to yield and may be completely exposed in a cavity, even when the circulation is still going on in them. Unfortunately, the erosion of a large vessel which has not yet been obliterated is by no means infrequent, and causes profuse and often fatal hæmorrhage. Another common event is the development of aneurisms on the arteries running in the walls of cavities. These may be small, bunch-like dilatations, or they may form cavities the size of a walnut or even larger. Rasmussen, Douglas Powell and others have called attention to their importance in hæmoptysis, under which section they are dealt with more fully.

And finally, about cavities of all sorts, the connective tissue develops and tends to limit the extent. The thickening is particularly marked beneath the pleura, and in chronic cases an entire apex may be converted into a mass of fibrous tissue, enclosing a few small cavities.

(*e*) *Pleura.*—Practically, in all cases of chronic phthisis the pleura is involved. Adhesions take place which may be thin and readily torn, or dense and firm, uniting layers of from two to five millimetres in thickness. This pleurisy may be simple, but in many cases it is tuberculous, and miliary tubercles or caseous masses are seen in the thickened pleural mem-

brane. Pleural effusion is not at all infrequent, either serous, purulent, or hæmorrhagic. Pneumothorax is a common accident.

(*f*) Changes in the *smaller bronchi* control the situation in the early stages of tuberculous phthisis, and play an important *rôle* throughout the disease. The process very often begins in the walls of the smaller tubes and leads to caseation, distention with products of inflammation, and broncho-pneumonia of the lobules. In many cases the visible implication of the bronchus is an extension upward of a process which has begun in the smallest bronchiole. This involvement weakens the wall, leading to bronchiectasis, not an uncommon event in phthisis. The mucous membrane of the larger bronchi, which is usually involved in a chronic catarrh, is more or less swollen, and in some instances ulcerated.

(*g*) The *bronchial glands*, in the more acute cases, are swollen and œdematous. Miliary tubercles and caseous foci are usually present. In cases of chronic phthisis the caseous areas are common, calcification may occur, and not infrequently purulent softening.

(*h*) *Changes in the other Organs.*—Of these, tuberculosis is the most common. In my series of autopsies the brain presented tuberculous lesions in 31, the spleen in 33, the liver in 12, the kidneys in 32, the intestines in 65, and the pericardium in 7. Other groups of lymphatic glands besides the bronchial may be affected—the cervical, the mediastinal, and the retro-peritoneal.

Certain degenerations are common. *Amyloid change* is frequent in the liver, spleen, kidneys, and mucous membrane of the intestines. The *liver* is often the seat of extensive fatty infiltration, which may cause marked enlargement. The *intestinal tuberculosis* occurs in advanced cases and is responsible in great part for the troublesome diarrhœa.

Endocarditis is not very uncommon, and was present in 12 of my post-mortems and in 27 of Percy Kidd's 500 cases. Tubercles may be present on the endocardium, particularly of the right ventricle. As pointed out by Norman Chevers and confirmed by subsequent writers, the subjects of congenital stenosis of the pulmonary orifice very frequently have phthisis.

The *larynx* is frequently involved, and ulceration of the vocal cords and destruction of the epiglottis are not at all uncommon.

Modes of Onset.—We have already seen that tuberculosis of the lungs may occur as the chief part of a general infection, or may set in with symptoms which closely simulate acute pneumonia. In the ordinary type of pulmonary tuberculosis the invasion is gradual and less striking, but presents an extraordinarily diverse picture, so that the practitioner is often led into error. Among the most characteristic of these types of onset are the following: (*a*) *With dyspeptic and anæmic symptoms*, forming a large and important group. The patients may naturally have had feeble digestion. They begin to show marked signs of dyspepsia and become pale, lose flesh, and look chlorotic before any pulmonary symptoms are

manifest. (*b*) *With chills and fever.* This mode of onset is particularly important in malarial regions, as the diagnosis of ordinary intermittent fever is often made, and the nature of the disease entirely overlooked. In Philadelphia it was very common to have patients sent to hospital supposed to be suffering with malaria, who had well-developed signs of pulmonary tuberculosis. (*c*) *Bronchitic onset.* These are the instances which arise in what the patient calls a neglected cold. The patient has perhaps been subject to naso-pharyngeal catarrh, and has been liable to take cold readily; then a bronchial cough develops, which proves intractable. Sometimes the bronchitic symptoms are associated with wheezing, like mild asthma. The development in these instances may be extremely insidious and, without any special aggravation of the general symptoms or increase in the fever, the tuberculous nature of the trouble may be discovered accidentally by the examination of the sputum. (*d*) *Onset with hæmoptysis.* The relation of hæmoptysis to pulmonary tuberculosis will be discussed elsewhere. The hæmoptysis may come on in a condition of robust health, and it occasionally, though rarely, happens that the pulmonary symptoms follow rapidly. In other cases a long interval elapses. Undoubtedly these are cases in which there has been a small localized lesion in the lung which has not produced constitutional disturbance. (*e*) *Pleuritic onset.* This may be a dry pleurisy, developing at the apex or in a scapular region, or in some instances extending generally. It may be acute pleurisy with effusion, or the effusion may have come on insidiously without any acute manifestations. Phthisis developed in a third of ninety cases of pleurisy with effusion, the subsequent history of which was followed by Bowditch. (*f*) *With laryngeal symptoms.* In rare instances huskiness and loss of voice are the symptoms for which the patient seeks advice, and the epiglottis or cords may be involved in a well-characterized tuberculosis before the physical signs in the lungs are at all clear. It is in these instances that the examination of the sputa is of the greatest value.

These represent the usual modes of onset of the ordinary chronic phthisis. It occasionally happens that in an instance with an acute pneumonic onset the severity of the symptoms subsides, and, instead of terminating as a majority of these cases do within ten or twelve weeks, the case drags on and becomes chronic.

Symptoms.—In discussing the symptoms it is usual to divide the disease into three periods: the first embracing the time of the growth and development of the tubercles; the second, in which they soften; and the third, in which there is a formation of cavities. Unfortunately, these anatomical stages can not be satisfactorily correlated with corresponding clinical periods, and we often find that a patient in the third stage with well-marked cavity is in a far better condition and has greater prospects of recovery than a patient in the first stage with diffuse consolidation. It is therefore better perhaps to disregard them altogether.

1. **Local Symptoms.**—*Pain* in the chest may be early and troublesome or absent throughout. It is usually associated with pleurisy, and may be sharp and stabbing in character, and either constant or felt only during coughing. Perhaps the commonest situation is in the lower thoracic zone, though in some instances it is beneath the scapula or referred to the apex. The attacks may recur at long intervals. Intercostal neuralgia occasionally develops in the course of ordinary phthisis.

Cough is one of the earliest symptoms, and is present in the majority of cases from beginning to end. There is nothing peculiar or distinctive about it. At first dry and hacking, and perhaps scarcely exciting the attention of the patient, it subsequently becomes looser, more constant, and associated with a glairy, muco-purulent expectoration. In the early stages of the disease the cough is bronchial in its origin. When cavities have formed it becomes more paroxysmal, and is most marked in the morning or after a sleep. Cough is not a constant symptom, however, and a patient may present himself with well-marked excavation at one apex who will declare that he has had little or no cough. So, too, there may be well-marked physical signs, dulness and moist sounds, without either expectoration or cough. In well-established cases the nocturnal paroxysms are most distressing and prevent sleep. The cough may be of such persistence and severity as to cause vomiting, and the patient becomes rapidly emaciated from loss of food.

Sputum.—This varies greatly in amount and character at the different stages of ordinary phthisis. There are cases with well-marked local signs at one apex, with slight cough and moderately high fever, without from day to day a trace of expectoration. So, also, there are instances with the most extensive consolidation (caseous pneumonia), with high fever, and, as in a recent instance under observation for several months, without enough expectoration to enable an examination for bacilli to be made. In the early stage of pulmonary tuberculosis the sputum is chiefly catarrhal and has a glairy, sago-like appearance, due to the presence of alveolar cells which have undergone the myelin degeneration. There is nothing distinctive or peculiar in this form of expectoration, which may persist for months without indicating serious trouble. The earliest trace of characteristic sputum is seen in the presence of small grayish or greenish-gray purulent masses. These, when coughed up, are always suggestive and should be the portions picked out for microscopical examination. As softening comes on, the expectoration becomes more profuse and purulent, but may still contain a considerable quantity of alveolar epithelium. Finally, when cavities exist, the sputa assume the so-called nummular form; each mass is isolated, flattened, greenish-gray in color, quite airless, and sinks to the bottom when spat into water.

By the microscopical examination of the sputum we determine whether the process is tuberculous, and whether softening has occurred. For *tubercle bacilli* the Ehrlich-Weigert method is the best. Eleven centimetres

of a saturated solution of fuchsin in absolute alcohol is added to one hundred centimetres of the saturated solution of commercial aniline oil (made by shaking up the oil in water and then filtering). This should be made fresh every third or fourth day. A small bit of the sputum is picked out on a needle or platinum wire and spread thin on the top-cover so as to make a uniformly thin layer. The top-cover is slowly dried about a foot above a Bunsen burner. Sufficient of the staining fluid is then dropped upon the top-cover, which is held at a little distance above the flame until the fluid boils. The staining fluid is then washed off in distilled water or put under the tap, decolorized in thirty per cent nitric-acid fluid, again washed off in water, and mounted on the slide. In doubtful cases the long process is used, the cover-slips remaining twenty-four hours in the stain. The bacilli are seen as elongated, slightly curved, red rods, sometimes presenting a beaded appearance. They are frequently in groups of three or four, but the number varies considerably. Only one or two may be found in a preparation, or, in some instances, they are so abundant that the entire field is occupied.

The presence of these bacilli in the sputum is an infallible indication of the existence of tuberculosis.

Sometimes they are found only after repeated examination. They may be abundant early in the disease and are usually numerous in the nummular sputum of the later stages.

Elastic tissue may be derived from the bronchi, the alveoli, or from arterial coats; and naturally the appearance of the tissue will vary with the locality from which it comes. In the examination for this it is not necessary to boil the sputum with caustic potash. For years I have used a simple plan which was shown to me at the London Hospital by Sir Andrew Clark. This method depends upon the fact that in almost all instances if the sputum is spread in a sufficiently thin layer the fragments of elastic tissue can be seen with the naked eye. The thick, purulent portions are placed upon a glass plate fifteen by fifteen centimetres and flattened into a thin layer by a second glass plate ten by ten centimetres. In this compressed grayish layer between the glass slips any fragments of elastic tissue show on a black background as grayish-yellow spots and can either be examined at once under a low power or the uppermost piece of glass is slid along until the fragment is exposed, when it is picked out and placed upon the ordinary microscopic slide. Fragments of bread and collections of milk-globules may also present an opaque white appearance, but with a little practice they can readily be recognized. Fragments of epithelium from the tongue, infiltrated with micrococci, are still more deceptive, but the microscope at once shows the difference.

The bronchial elastic tissue forms an elongated network, or two or three long, narrow fibres are found close together. From the blood-vessels a somewhat similar form may be seen and occasionally a distinct

sheeting is found as if it had come from the intima of a good-sized artery. The elastic tissue of the alveolar wall is quite distinctive; the fibres are branched and often show the outline of the arrangement of the air cells. The elastic tissue from bronchus or alveoli indicates extensive erosion of a tube and softening of the lung-tissue.

Another occasional constituent of the sputum is blood, which may be present as the chief constituent of the expectoration in hæmoptysis or may simply tinge the sputum. In chronic cases with large cavities, in addition to bacteria, various forms of fungi may develop, of which the aspergillus is the most important. Sarcinæ may also occur.

The daily amount of expectoration varies. In rapidly advancing cases, with much cough, it may reach as high as five hundred cubic centimetres in the day. In cases with large cavities the chief amount is brought up in the morning. The expectoration of tuberculous patients usually has a heavy, sweetish odor, and occasionally it is fetid, owing to decomposition in the cavities.

Hæmoptysis.—Hæmoptysis is met with either early in the disease, before there are physical signs, or during the course of the affection when there is softening or excavation. A majority of the hæmorrhages believed to be precursory are really due to already existing disease of the lung, and there is no ground whatever for the opinion, so long held, that phthisis can originate directly from hæmoptysis. The blood may be either pure or mixed with sputum. A distinction should be made between these two forms. When the sputa are simply tinged or the blood is admixed, it comes, in all probability, from hyperæmic bronchial mucosa or locally congested areas of lung-tissue; but the brisk hæmorrhage in which the blood comes up in mouthfuls is always due to erosion of vessels, small or large, in the process of softening, or, in the later stages of the disease, comes from the erosion of a branch of the pulmonary artery or from a ruptured aneurism of the pulmonary artery in a cavity. This latter is the most frequent cause of the fatal hæmorrhage in consumption.

Dyspnœa is not a common accompaniment of ordinary phthisis. The greater part of one lung may be diseased and local trouble exist at the other apex without any shortness of breath. Even in the paroxysms of very high fever the respirations may not be much increased. Rapid advance, as of a broncho-pneumonic process, or the development of miliary tubercles throughout the lung, causes great increase in the number of respirations. A degree of dyspnœa leading to cyanosis is almost unknown, apart from extensive invasion of the sound portions by miliary tubercles. One reason why there is so little shortness of breath in phthisis is that there is always a moderate grade of anæmia, and the diminished lung-space is sufficient to supply oxygen to the reduced number of blood-corpuscles.

2. **General Symptoms.**—*Fever.*—To get a correct idea of the temperature range in pulmonary tuberculosis it is necessary, as Ringer pointed

out, to make tolerably frequent observations. The usual 8 A. M. and 8 P. M. record is, in a majority of the cases, very deceptive, giving neither the minimum nor maximum. The former usually occurs between 2 and 6 A. M. and the latter between 2 and 6 P. M.

A recognition of various forms of fever, viz., of tuberculization, of ulceration, and of absorption, emphasizes the anatomical stages of growth, softening and cavity formation; but practically such a division is of little use, as in a majority of cases these processes are going on together.

Fever is the most important initial symptom and throughout the entire course the thermometer is the most trustworthy guide as to the progress of the affection. With pyrexia a patient loses in weight and strength, and the local disease usually progresses. The periods of apyrexia are those of gain in weight and strength and in limitation of the local lesion. It by no means necessarily follows that a patient with tuberculosis has pyrexia. There may be quite extensive disease without coexisting fever. At the moment of writing, I have eighteen instances of chronic phthisis under observation, of whom ten are practically free from fever; but in the early stage, when tubercles are developing and caseous areas are in process of formation and when softening is in progress, fever is a constant symptom. It was present in one hundred consecutive cases at my dispensary service.

Two types of fever are seen—the remittent and the intermittent. These may occur indifferently in the early or in the late stages of the disease or may alternate with each other, a variability which depends upon the fact that phthisis is a progressive disease and that all stages of lesions may be found in a single lung. Special stress should be laid upon the fact, particularly in malarial regions, that tuberculosis may set in with a fever typically intermittent in character—a daily chill, with subsequent fever and sweat. In Montreal, where malaria is practically unknown, this was always regarded as a suggestive symptom; but in Philadelphia and Baltimore, where ague prevails, it is no exaggeration to say that yearly scores of cases of early tuberculosis are treated for ague. These are often cases that pursue a rapid course. The fever of onset—tuberculization—may be almost continuous, with slight daily exacerbations; and at any time during the course of chronic phthisis, if there is rapid extension, the remissions become less marked.

A remittent fever, in which the temperature is constantly above normal but drops two or three degrees toward morning, is not uncommon in the middle and later stages and is usually associated with softening or extension of the disease. Here, too, a simple morning and evening register may give an entirely erroneous idea as to the range of the fever. With breaking down of the lung-tissue and formation of cavities, associated as these processes always are with suppuration and with more or less systemic contamination, the fever assumes a characteristically intermittent or hectic type. For a large part of the day the patient is not only afebrile,

but the temperature is subnormal. In the annexed two-hourly chart, from a case of chronic tuberculosis of the lungs, it will be seen that from 10 P. M. to 8 or 12 A. M., the temperature continuously fell and reached as low as 95°. A slow rise then took place through the late morning and early afternoon hours and reached its maximum between 6 and 10 P. M. As shown in the chart there were in the three days about forty-three hours of pyrexia and twenty-nine hours of apyrexia. The rapid fall of

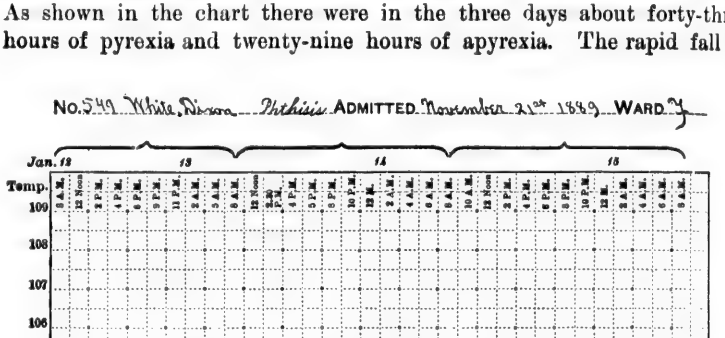


CHART XIII.—Three days. Chronic tuberculosis.

the temperature in the early morning hours is usually associated with sweating. This hectic, as it is called, which is a typical fever of septic infection, is met with when the process of cavity formation and softening is advanced and extending.

A continuous fever with remissions of not more than a degree, developing in the course of pulmonary tuberculosis, is suggestive of acute pneumonia. When a two-hourly chart is made, the remissions even in acute tuberculous pneumonia are usually well marked. A continued fever, such

as is seen in the first week of typhoid, or in some cases of inflammation of the lung, is rare in tuberculosis.

Sweating.—Drenching perspirations are common in phthisis and constitute one of the most distressing features of the disease. They occur usually at night, or at any time in the day when the patient sleeps. They may come on early in the disease, but are more persistent and frequent after cavities have formed. Some patients escape altogether.

The *pulse* is increased in frequency, especially when the fever is high. It is often remarkably full, though soft and compressible. Pulsation may sometimes be seen in the capillaries and in the veins on the back of the hand.

Emaciation is a pronounced feature. The loss of weight is gradual but, if the disease is extending, progressive. The scales give one of the best indications of the progress of the case.

3. **Physical Signs.**—(*a*) *Inspection.*—The shape of the chest is often suggestive, though it is to be remembered that pulmonary tuberculosis may be met with in chests of any build. Practically, however, in a considerable proportion of cases the thorax is long and narrow, with very wide intercostal spaces, the ribs more vertical in direction and the costal angle very narrow. The scapulæ are "winged," a point noted by Hippocrates. Another type of chest which is very common is that which is flattened in the antero-posterior diameter. The costal cartilages may be prominent and the sternum depressed. Occasionally the lower sternum forms a deep concavity, the so-called funnel breast (*Trichter-Brust*). Inspection gives valuable information in all stages of the disease. Special examination should be made of the clavicular regions to see if one clavicle stands out more distinctly than the other, or if the spaces above or below it are more marked. Defective expansion at one apex is an early and important sign. The condition of expansion of the lower zone of the thorax may be well estimated by inspection. The condition of the præcordia should also be noted, as a wide area of impulse, particularly in the second, third, and fourth interspaces, often results from disease of the left apex. From a point behind the patient, looking over the shoulders, one can often better estimate the relative expansion of the apices.

(*b*) *Palpation.*—Deficiency in expansion at the apices or bases is perhaps best gauged by placing the hands in the subclavicular spaces and then in the lateral regions of the chest and asking the patient to draw slowly a full breath. Standing behind the patient and placing the thumbs in the supraclavicular and the fingers in the infraclavicular spaces one can judge accurately as to the relative mobility of the two sides. Disease at an apex, though early and before dulness is at all marked, may be indicated by deficient expansion. On asking the patient to count, the tactile fremitus is increased wherever there is local growth of tubercle or extensive caseation. In comparing the apices it is important to bear in mind that normally the fremitus is stronger at the right than

at the left. So too at the base, when there is consolidation of the lung, the fremitus is increased; whereas, if there is pleural effusion, it is diminished or absent. In the later stages, when cavities form, the tactile fremitus is usually much exaggerated over them. When the pleura is greatly thickened the fremitus may be somewhat diminished.

(*c*) *Percussion.*—Tubercles, inflammatory products, fibroid changes, and cavities produce important changes in the pulmonary resonance. There may be localized disease, even of some extent, without inducing much alteration; as when the tubercles are scattered and have air-containing tissue between them. One of the earliest and most valuable signs is defective resonance upon and above a clavicle. In a considerable proportion of all cases of phthisis the dulness is first noted in these regions. The comparison between the two sides should be made also when the breath is held after a full inspiration, as the defective resonance may then be more clearly marked. In the early stages the percussion note is usually higher in pitch and may require an experienced ear to detect the difference. In recent consolidation from caseous pneumonia the percussion note often has a tubular or tympanitic quality. A wooden dulness is rarely heard except in old cases with extensive fibroid change at the apex or base. Over large, thin-walled cavities at the apex the so-called cracked-pot sound may be obtained. In thin subjects the percussion should be carefully practised in the supraspinous fossæ and the interscapular space, as they correspond to very important areas early involved in the disease. In cases with numerous separated cavities at the apex, without much fibroid tissue or thickening of the pleura, the percussion note may show little change, and the contrast between the signs obtained on auscultation and percussion is most marked.

(*d*) *Auscultation.*—Feeble breath-sounds are among the most characteristic early signs, since not as much air enters the tubes and vesicles of the affected area. It is well at first always to compare carefully the corresponding points on the two sides of the chest without asking the patient either to draw a deep breath or to cough. With early apical disease the inspiration on quiet breathing may be scarcely audible. Expiration is usually prolonged. On the other hand there are cases in which the earliest sign is a harsh, rude, respiratory murmur. On deep breathing it is frequently to be noted that inspiration is jerking or wavy, the so-called "cog-wheel" rhythm; which, however, is by no means confined to tuberculosis. With extension of the disease the inspiratory murmur is harsh, and, when consolidation occurs, whiffing and bronchial. With these changes in the character of the murmur there are râles, due to the accompanying bronchitis. They may be heard only on deep inspiration or on coughing, and early in the disease are often crackling in character. When softening occurs they are louder and have a bubbling, sometimes a characteristic clicking quality. These "moist sounds," as they are called, when associated with change in the percussion resonance are extremely suggestive.

When cavities form, the râles are louder, more gurgling, and resonant in quality. When there is consolidation of any extent the breath-sounds are tubular, and in the large excavations loud and cavernous, or have an amphoric quality. In the unaffected portions of the lobe and in the opposite lung the breath-sounds may be harsh and even puerile. The vocal resonance is usually increased in all stages of the process, and bronchophony and pectoriloquy are met with in the regions of consolidation and over cavities. Pleuritic friction may be present at any stage and, as mentioned before, occurs very early. There are cases in which it is a marked feature throughout. When the lappet of lung over the heart is involved there may be a pleuro-pericardial friction, and when this area is consolidated there may be curious clicking râles synchronous with the heart-beat, due to the compression by the heart of, and the expulsion of air from, this portion. An interesting auscultatory sign, met most commonly in phthisis, is the so-called cardio-respiratory murmur, a whiffing systolic bruit due to the propulsion of air out of the tubes by the impulse of the heart. It is best heard during inspiration and in the antero-lateral regions of the chest.

A systolic murmur is frequently heard in the subclavian artery on either side, the pulsation of which may be very visible. The murmur is in all probability due to pressure on the vessels by the thickened pleura.

The signs of cavity may be here briefly enumerated.

(*a*) When there is not much thickening of the pleura or condensation of the surrounding lung-tissue, the percussion sound may be full and clear, resembling the normal note. More commonly there is defective resonance or a tympanitic quality which may at times be purely amphoric. The pitch of the percussion note changes over a cavity when the mouth is opened or closed (Wintrich's sign), or it may be brought out more clearly on change of position. The cracked-pot sound is only obtainable over tolerably large cavities with thin walls. It is best elicited by a firm, quick stroke, the patient at the time having the mouth open. In those rare instances of almost total excavation of one lung the percussion note may be amphoric in quality. (*b*) On auscultation the so-called cavernous sounds are heard: (1) Various grades of modified breathing—blowing or tubular, cavernous or amphoric. There may be a curiously sharp hissing sound, as if the air was passing from a narrow opening into a wide space. In very large cavities both inspiration and expiration may be typically amphoric. (2) There are coarse bubbling râles which have a resonant quality, and on coughing may have a metallic or ringing character. On coughing they are often loud and gurgling. In very large thin-walled cavities, and more rarely in medium-sized cavities, surrounded by recent consolidation, the râles may have a distinctly amphoric echo, simulating those of pneumothorax. There are dry cavities in which no râles are heard. (3) The vocal resonance is greatly intensified and whispered pectoriloquy is clearly heard. In large apical cavities the heart-sounds are well heard, and occasionally there may be an intense systolic murmur,

probably always transmitted to, and not produced, as has been supposed, in the cavity itself.

Pseudo-cavernous signs may be caused by an area of consolidation near a large bronchus. The condition may be most deceptive—the high-pitched or tympanic percussion note, the tubular or cavernous breathing, and the resonant râles, simulate closely those of cavity.

4. **Symptoms referable to other Organs.**—(*a*) *Cardio-vascular.*—The retraction of the left upper lobe exposes a large area of the heart. In thin-chested subjects there may be pulsation in the second, third, and fourth interspaces close to the sternum. Sometimes with much retraction of the left upper lobe the heart is drawn up. A systolic murmur over the pulmonary area is common in all stages of phthisis. Apical murmurs are also not infrequent and may be extremely rough and harsh without necessarily indicating that endocarditis is present. The association of heart-disease with phthisis is not, however, very uncommon. As already mentioned, there were twelve instances of endocarditis in 216 autopsies. The arterial tension is usually low in phthisis and the capillary resistance lessened so that the pulse is often full and soft even in the later stages of the disease. The capillary pulse is not infrequently met with, and pulsation of the veins in the back of the hand is occasionally to be seen.

(*b*) *Blood Glandular System.*—The early anæmia has already been noted. It is often more apparent than real, a chloro-anæmia, and the blood-count rarely sinks below two million per cubic millimetre.

The blood-plates are, as a rule, enormously increased and are seen in the withdrawn blood as the so-called Schultze's granule masses.

(*c*) *Gastro-intestinal System.*—The tongue is usually furred, but may be clean and red. Small aphthous ulcers are sometimes distressing. A red line on the gums, a symptom to which at one time much attention was paid as a special feature of phthisis, occurs in other cachectic states. Extensive tuberculous disease of the pharynx, associated with similar affection of the larynx, may interfere seriously with deglutition and prove a very distressing and intractable symptom.

Of late, special attention has been paid to the gastric symptoms of this affection. Tuberculous disease is rare. I have seen but one undoubted specimen from a case which Musser has reported.* Ulceration may occur as an accidental complication and multiple catarrhal ulcers are not uncommon. Interstitial and parenchymatous changes in the mucosa are common (possibly associated with the venous stasis) and lead to atrophy, but these cannot always be connoted with the symptoms, and they may be found when not expected. On the other hand, when the gastric symptoms have been most persistent, the mucosa may show very little change. It is impossible always to refer the anorexia, nausea, and vomiting of consumption to local conditions. The hectic fever and the neurotic influ-

* Philadelphia Hospital Reports, vol. i, 1890.

ences, upon which Immermann lays much stress, must be taken into account, as they play an important *rôle*. The organ is often dilated, and to muscular insufficiency alone may be due some of the cases of dyspepsia. The condition of the gastric secretion is not constant, and the reports are discordant. In the early stages there may be hyperacidity; later, a deficiency of acid.

Anorexia is often a marked symptom at the onset; there may be positive loathing of food, and even small quantities cause nausea. Sometimes without any nausea or distress after eating the feeding of the patient is a daily battle. When practicable, Debove's forced alimentation is of great benefi in such cases. Nausea and vomiting, though occasionally troublesome at an early period, are more marked in the later stages. The latter may be caused by the severe attacks of coughing. S. H. Habershon refers to four different causes the vomiting in phthisis: (1) central, as from tuberculous meningitis; (2) pressure on the vagi by caseous glands; (3) stimulation from the peripheral branches of the vagus, either pulmonary, pharyngeal, or gastric; and (4) mechanical causes.

Of the *intestinal* symptoms diarrhœa is the most serious. It may come on early, but is more usually a symptom of the later stages, and is associated with ulceration, particularly of the large bowel. Extensive ulceration of the ileum may exist without any diarrhœa. The associated catarrhal condition may account in part for it, and in some instances the amyloid degeneration of the mucous membrane.

(*d*) *Nervous System.*—(1) Focal lesions due to the development of coarse tubercles and areas of tuberculous meningo-encephalitis. Aphasia, for instance, may result from the growth of meningeal tubercles in the fissure of Sylvius, or even hemiplegia may develop. The solitary tubercles are more common in the chronic phthisis of children. (2) Basilar meningitis is an occasional complication. It may be confined to the brain, though more commonly it is a (3) cerebro-spinal meningitis, which may come on in persons without well-developed local signs in the chest. Twice have I known strong, robust men brought into hospital with signs of cerebro-spinal meningitis, in whom the existence of pulmonary disease was not discovered until the post-mortem. (4) Peripheral neuritis. This is not frequent, and has occurred but five times in the large number of consumptives who have come under my observation during the past seven years. It is nearly always an extensor paralysis of the arm or leg, more commonly the latter, causing foot-drop. It is usually a late manifestation. (5) Mental symptoms. It was noted, even by the older writers, that consumptives had a peculiarly hopeful temperament, and the *spes phthisica* forms a curious characteristic of the disease. Patients with extensive cavities, high fever, and too weak to move will often make plans for the future and confidently expect to recover.

Apart from tuberculosis of the brain, there is sometimes in chronic phthisis a form of insanity not unlike that which develops in the conva-

lescence from acute affections. The whole question of the mutual relations of insanity and phthisis is dealt with at length in Mickle's Gulstonian lectures.

(*e*) A remarkable hypertrophy of the mammary gland may occur in pulmonary tuberculosis,* most commonly in males. It may only be on the affected side. Two cases came under my notice at the University Hospital, Philadelphia, both in young males. It is a chronic interstitial, non-tuberculous mammitis (Allot).

(*f*) *Genito-urinary System.*—The urine presents no special peculiarities in amount or constituents. Fever, however, has a marked influence upon it. Albumen is met with frequently and may be associated with the fever, or is the result of definite changes in the kidneys. In the latter case it is more abundant and more curd-like. Amyloid disease of the kidneys is not uncommon. Its presence is shown by albumen and tube-casts in the urine, and sometimes by a great increase in the amount of urine. In other instances there is dropsy, and the patients have all the characteristic features of chronic Bright's disease.

Pus in the urine may be due to disease of the bladder or of the pelves of the kidneys. In some instances the entire urinary tract is involved. In pulmonary phthisis, however, extensive tuberculous disease is rarely found in the urinary organs. Bacilli may occasionally be detected in the pus. Hæmaturia is not a very common symptom. It may occur occasionally as a result of congestion of the kidneys, which passes off and leaves the urine albuminous. In other instances it results from disease of the pelvis or of the bladder, and is associated either with early tuberculosis of the mucous membranes or more commonly with ulceration.

(*g*) *Cutaneous System.*—The skin is often dry and harsh. Local tubercles occasionally develop on the hands. There may be pigmentary staining, the *chloasma phthisicorum*, which is more common when the peritonæum is involved. Upon the chest and back the brown stains of the *pityriasis versicolor* are very frequent. The hair of the head and beard may become dry and lanky. The terminal phalanges, in chronic cases, become clubbed and the nails incurvated—the Hippocratic fingers.† A remarkable and unusual complication is general emphysema, which may result from ulceration of an adherent lung or perforation of the larynx.

Diagnosis.—When well advanced there is rarely any doubt as to the existence of tuberculous phthisis, for the sputum gives positive information, and the physical signs of local disease are well marked. The bacilli give an infallible indication of the existence of tuberculosis and may be found in the sputum before the physical signs are at all definite. On the other hand, it must be remembered that there are cases in which, even

* Allot, Paris Thesis, 1887.

† "Morbo progrediente, corpus macrescit præter crura: hæc autem tument et pedes, et ungues contorquentur" (Hippocrates).

with tolerably well-defined physical signs, the sputum is extremely scanty and many examinations may be required to detect tubercle bacilli. So essential is the examination of the sputum in the early diagnosis of phthisis that I would earnestly insist upon the more frequent employment of this method. There is no excuse now for its omission, since, if the practitioner has not command of the necessary technique, there are laboratories in many parts of the country at which the examination can be made. *Early detection is of vital importance, as successful treatment depends upon the measures taken before the lung is extensively involved.*

The presence of elastic fibres in the sputum is an indication of destruction of the lung-tissue. In a large proportion of cases it is indicative, too, of tuberculous disease. It also may be found early, before the physical signs are well marked. Its detection is easy by the above-mentioned method, not requiring high powers of the microscope. In cases of early hæmoptysis, before there is marked constitutional disturbance, or even local signs, it is very important to make a thorough examination of the sputum, from which mucoid and purulent portions may be picked out for examination. With localized and persistent signs in one lung, cough, fever, and loss of flesh, the diagnosis is rarely dubious. It is remarkable, however, to what an extent the local process may sometimes proceed without disturbance of health sufficient to excite the alarm of the physician or friends. There are puzzling cases with localized physical signs at one apex, chiefly moist râles, rarely any percussion changes, perhaps slight fever, and a glairy expectoration containing numerous alveolar cells. I have seen several cases of this kind which have been for a time very obscure, and in which repeated examinations failed to detect either bacilli or elastic tissue. They seem to be instances of local catarrhal trouble in the smaller tubes, some of which clear in a few weeks.

3. Fibroid Phthisis.

In the section on diseases of the lungs we shall refer to the chronic interstitial pneumonia, or cirrhosis of the lung, which may be a sequence of acute lobar pneumonia, or follow a chronic pleurisy, or is due to inhalation of dust, as in anthracosis. From these causes a condition of sclerosis or induration of the lung may be produced with gradual shrinkage. An identical condition is present in certain cases of chronic pulmonary tuberculosis, and to this it is best perhaps to limit the term *fibroid phthisis*. This form may come on gradually as a sequence of a chronic tuberculous broncho-pneumonia, or follow a chronic tuberculous pleurisy. In other instances the process supervenes upon an ordinary ulcerative phthisis. The disease becomes limited to one apex, the cavity is surrounded by layers of dense fibrous tissue, the pleura is thickened, and the lower lobe is gradually invaded by the sclerotic change. Ultimately a picture is produced little if at all different from the other forms of cirrhosis of the

lungs. It may even be difficult to say that the process is tuberculous, but in advanced cases the bacilli are usually present in the walls of the cavity at the apex, or old, encapsulated caseous areas exist in the lung, or there may be tubercles at the apex of the other lung and in the bronchial glands. Dilatation of the bronchi is present; the right ventricle, sometimes the entire heart, is hypertrophied.

The disease is chronic, lasting from ten to twenty or more years, during which time the patient may have fair health.

The chief symptoms are cough, which is often paroxysmal in character and most marked in the morning. The expectoration is purulent, and in some instances, when the bronchiectasis is extensive, fœtid. There is dyspnœa on exertion, but little or no fever.

The physical signs are very characteristic. The chest is sunken and the shoulder lower on the affected side; the heart is often drawn over and displaced. If the left lung is involved there may be an unusually large area of cardiac pulsation in the third, fourth, and fifth interspaces. Heart-murmurs are common. There is dulness over the affected side and deficient tactile fremitus. At the apex there may be well-marked cavernous sounds; at the base, distant bronchial breathing. The condition may persist indefinitely. In some cases the other lung becomes involved, or the patient has repeated attacks of hæmoptysis, in one of which he dies. As a result of the chronic suppuration, amyloid degeneration of the liver, spleen, and intestines may take place; dropsy frequently supervenes from failure of the right heart.

A more detailed account is found under Cirrhosis of the Lung, with which this form is clinically identical.

Diseases associated with Pulmonary Tuberculosis.

Lobar pneumonia is not an uncommon cause of death. It is met with most frequently, indeed, as a terminal event in the chronic cases. It may, however, occur early, and be difficult to distinguish from an acute caseous pneumonia. The sputa in the latter are rarely rusty, while the fever in the former is more continuous and higher, but in many cases it is impossible to differentiate between the two conditions.

Typhoid fever is rare in phthisis, but cases unquestionably occur. In *Case 8* of my series of post-mortems in this disease, a girl, aged eighteen, had peritoneal adhesions, local disease at both apices, and perfectly characteristic enteric lesions. In *Case 34*, a male, aged twenty-five, with tuberculous cavities, had a very acute attack. The Peyer's glands were greatly swollen with adherent sloughs. The spleen weighed 533 grammes. The characters of the ulceration are usually distinctive.

Erysipelas not infrequently attacks old *poitrinaires* in hospital wards and almshouses. There are instances in which the attack seems to be beneficial, as the cough lessens and the symptoms ameliorate. It may, however, prove fatal, as in a recent case admitted to my wards.

The *eruptive fevers*, particularly measles, frequently precede, but rarely develop in the course of pulmonary tuberculosis. In the revaccination of a tuberculous subject the vesicles run a normal course.

Fistula in ano is associated with phthisis in an interesting manner. In a majority of such cases it is a tuberculous process. The general affection may progress rapidly after an operation. The question is considered in tuberculosis of the alimentary canal.

In chronic and arrested phthisis *arterio-sclerosis* is not uncommon. Ormerod noted thirty cases of chronic renal disease in one hundred post mortems.

The association of tuberculosis with *chronic arthritis*, upon which certain writers lay stress, finds its explanation in the lowered resistance of these patients, and the greater liability to infection in the institutions in which so many of them live.

Peculiarities of Pulmonary Tuberculosis at the Extremes of Life.

(*a*) *Old Age.*—It is remarkable how common tuberculosis is in the aged, particularly in institutions. McLachlan noted a hundred and forty-five cases in which tuberculosis was the cause of death in old persons in Chelsea Hospital. All were over sixty years of age. The experience at Salpêtrière is the same. Laennec met with a case in a person over ninety-nine years of age.

At the Philadelphia Hospital, in the bodies of aged persons sent over from the almshouse it was extremely common to find either old or recent tuberculosis. A patient died under my care at the age of eighty-two with extensive peritoneal tuberculosis. Pulmonary tuberculosis in the aged is usually latent and runs a slow course. The physical signs are often masked by emphysema and by the coexisting chronic bronchitis. The diagnosis may depend entirely upon the discovery of the bacilli and elastic tissue. Contrary to the opinion which was held some years ago, tuberculosis is by no means uncommon with senile emphysema. Some of the cases of tuberculosis in the aged are instances of quiescent disease which may have dated from an early period.

(*b*) *Infants.*—The occurrence of acute tuberculosis in children has already been mentioned, and also the fact that the disease is occasionally congenital. Recent studies, particularly of French writers, have shown that it is a frequent affection in children under two years of age. Leroux has analyzed the statistics of the late Prof. Parrot, embracing 219 cases in children under three years. Of these there were from one day to three months, 23; from three to six months, 35; from six to twelve months, 53 (a total of 111 under one year); and from one to two years, 108. Pulmonary cavities were present in 57 of the cases, and in only 50 was the pulmonary lesion the sole manifestation. At the St. Petersburg Foundling Asylum, in the ten years ending 1884, there were 416 cases of tuberculosis in 16,581 autopsies. The observations of Northrup, at the New

York Foundling Hospital, are of special interest in connection with the mode of infection. Of 125 cases of tuberculosis on the records of this institution, in 34 the ravages were extensive, the seat of the primary affection was not clear, and the bronchial glands were large and cheesy. In 20 cases of general tuberculosis there were cheesy masses in the bronchial glands and in the lungs. In 42 cases of general tuberculosis the only cheesy masses were in the bronchial lymph-glands. In 9 cases the tubercles were limited to the bronchial nodes and the lungs; the latter containing only discrete miliary bodies, while the bronchial glands were in advanced caseation. In 13 cases there was tuberculosis of the bronchial nodes only. In most of these cases the patients died of infectious diseases. These figures are very suggestive, and point, as already noted, to infection through the bronchial passages as the most common method, even in children. Of 500 autopsies in children at the Munich Pathological Institute, in 150 (thirty per cent) tuberculosis was present and in over ninety-two per cent the lungs were involved (Müller).

Modes of Death in Pulmonary Tuberculosis.

(*a*) *By asthenia*, a gradual failure of the strength. The end is usually peaceable and quiet,* occasionally disturbed by paroxysms of cough. Consciousness is often retained until near the close.

(*b*) *By asphyxia*, as in some cases of acute miliary tuberculosis and in acute pneumonic phthisis. In chronic phthisis it is rarely seen, even when pneumothorax develops.

(*c*) *By syncope.* This is not common. I have known it to happen once or twice in patients who insisted upon going about when in the advanced stages of the disease. There may be, but not necessarily, fatty degeneration of the heart. A rapidly developing syncope may follow hæmorrhage or may be due to thrombosis or embolism of the pulmonary artery, or to pneumothorax.

(*d*) *From hæmorrhage.* The fatal bleeding in chronic phthisis is due to erosion of a large vessel or rupture of an aneurism in the pulmonary cavity, most commonly the latter. Of twenty-six analyzed by S. West, in eleven cases the fatal hæmoptysis was due to aneurism, and of thirty-five cases collected by Percy Kidd, aneurism was present in thirty. In a case of Curtin's, at the Philadelphia Hospital, the bleeding proved fatal before hæmoptysis occurred, as the eroded vessel opened into a capacious cavity.

(*e*) *With cerebral symptoms.* Coma may be due to meningitis, less often to uræmia. Death in convulsions is rare. The hæmorrhagic pachymeningitis which develops in some cases of phthisis occasionally causes loss of consciousness, but is rarely a direct cause of death. In one of my

* As is so well described by Sir Thomas Browne, whose *Letter to a Friend* gives a unique account of the last illness of a consumptive. Hood's *Death-bed* is true of phthisis more frequently than of any other disease.

cases, death resulted from thrombosis of the cerebral sinuses with symptoms of meningitis.

V. Tuberculosis of the Serous Membranes.

General Pleuro-peritoneal Tuberculosis.—There are interesting cases in which the lesions are confined almost entirely to the serous sacs—the pleura, pericardium, and peritonæum. I do not here refer to instances of chronic pulmonary tuberculosis, in which the pleura and the peritonæum may be involved, but to the primitive form, in which these serous membranes are involved in either (*a*) *an acute miliary inflammation;* (*b*) *a chronic ulcerative tuberculosis;* or (*c*) *a chronic fibroid tuberculosis.* It is sufficient to indicate here the fact that cases occur involving primarily the pleura and peritonæum alone, sometimes with the pericardium, and to pass on to the consideration of the affections of the individual sacs.

Tuberculosis of the Pleura.—This may be primary or secondary.

Primary tuberculosis of the pleura occurs as an *acute* process associated with a sero-fibrinous or hæmorrhagic exudate. Unquestionably many of the cases regarded as pleurisy from cold are of this nature. It may be truly primary, but in many instances local tuberculous disease exists in lung or lymph-glands. There is a primary chronic tuberculosis of the pleura. This produces great thickening and caseation of both layers, which are separated from each other by a thin infiltrated connective tissue, in which miliary granulations may sometimes be seen. The pleural layers together may have a thickness of from five to ten millimetres. It is a comparatively rare affection. I found one of the most striking illustrations of the kind in a young, remarkably healthy-looking Irish girl, who died under my care of malignant scarlet fever. There were no other tuberculous lesions in the body. The condition may be unilateral or bilateral.

Secondary tuberculous pleurisy is very common. The visceral layer is always involved in pulmonary tuberculosis. Adhesions usually form and a chronic pleurisy results, which may be simple, but usually tubercles are scattered through the adhesions. An acute tuberculous pleurisy may result from direct extension. The fluid may be sero-fibrinous or hæmorrhagic, or may become purulent. And, lastly, a very common event in pulmonary tuberculosis is the perforation of a superficial spot of softening, and the production of *pyo-pneumothorax.*

The general symptomatology of these forms will be considered under diseases of the pleura.

Tuberculosis of the Pericardium.—Miliary tubercles may occur as a part of a general infection, but the term is properly limited to those cases in which, either as a primary or secondary process, there is extensive disease of the membrane. Tuberculosis is not so common in the pericardium as in the pleura and peritonæum, but it is certainly more com-

mon than the literature would lead us to suppose. Only thirty references are noted in the Index Catalogue of the Surgeon-General's Office. It occurs in two forms—chronic and acute.

(*a*) *Chronic Tuberculous Pericarditis.*—This may occur as a primary affection associated only with the caseation of the bronchial or particularly the anterior mediastinal lymph-glands. More commonly there is tuberculous disease elsewhere, either of the pleura or of the lungs, sometimes of the peritonæum. In a number of cases the pericarditis is only a part of a general infection of the serous membranes. The instances are very rare in which the process is confined to the pericardium. In one of my cases, a man aged seventy-two, who died of pneumonia in the Philadelphia Hospital, the pericardium was thickened, both leaves were adherent and presented cheesy masses and gray nodules. The heart weighed 554 grammes; the bronchial glands were calcified; there were no tubercles in the other organs. The disease occurs at all ages. My youngest case was in a child, aged five, in whom both layers of the pericardium were greatly thickened and cheesy. In nearly every instance the bronchial or mediastinal glands are tuberculous. Occasionally it is due to extension from tuberculous disease of the sternum or of the spine; occasionally to extension from the lungs. In one case, a man, aged fifty, who died in the Philadelphia Hospital, the outer layer of the pericardium was alone involved and thickened, in connection with a tuberculous abscess in the anterior mediastinum. The condition is usually unsuspected. The physical signs are those of hypertrophy of the heart. In a recent case the organ weighed 600 grammes, and the clinical symptoms were those of hypertrophy and dilatation.

The *physical signs* are somewhat uncertain, since they are those of adherent pericardium. The dulness may reach high along the left sternal margin, and in one case, in which it was as high as the middle of the manubrium, the thickened pericardial layers formed a solid cheesy mass which surrounded the aorta.

(*b*) *Acute Tuberculous Pericarditis.*—This may occur as a secondary infection from tubercle in other parts, or it may arise by direct extension from the lungs, or more commonly by invasion from mediastinal lymph-glands. The exudation may be limited in amount and chiefly fibrinous, or it may be serous, and in many cases is hæmorrhagic. Unless carefully sought for, the tubercles may be overlooked. Lastly, some of the cases of purulent pericarditis are tuberculous. The membranes may be much thickened and no trace of tubercles apparent. The nature of the case may, then, be gathered chiefly from the existence of tuberculous bronchial or mediastinal glands, or the existence of tuberculous foci in other regions. The effusion in these cases may be enormous, as in one reported by Musser, in which the sac contained sixty-four ounces of fluid.

The symptoms and physical signs of this condition will be considered with those of ordinary pericarditis with effusion.

(*d*) **Tuberculosis of the Peritonæum.**—In connection with miliary and chronic pulmonary tuberculosis it is not uncommon to find the peritonæum studded with small gray granulations. They are constantly present on the serous surface of tuberculous ulcers of the intestines. Apart from these conditions the membrane is often the seat of extensive tuberculous disease, which occurs in the following forms:

(1) *Acute miliary tuberculosis* with sero-fibrinous or bloody exudation.

(2) *Chronic tuberculosis*, characterized by larger growths, which tend to caseate and ulcerate. It may lead to perforation of the intestinal coils. The exudate is purulent or sero-purulent, and is often sacculated.

(3) *Chronic fibroid tuberculosis*, which may be subacute from the onset, or which may represent the final stage of an acute miliary eruption. The tubercles are hard and pigmented. There is little or no exudation, and the serous surfaces are matted together by adhesions.

The process may be primary and local, which was the case in five of my seventeen post-mortems. In children the infection appears to pass from the intestines, and in adults this is the source in the cases associated with chronic phthisis. In women the disease extends commonly from the Fallopian tubes. In at least 30 or 40 per cent of the instances of laparotomy in this affection reported by gynæcologists the infection was from them. The prostate or the seminal vesicles may be the starting-point. In many cases the peritonæum is involved with the pleura and pericardium, particularly with the former membrane.

It is generally stated that males are attacked oftener than females. In my own series of 21 cases, 15 were males. The recent laparotomies, however, which have been performed in this disease have been chiefly in females; so that in the collected statistics I find the cases to be twice as numerous in females as in males; in the ratio, indeed, of 131 to 60.

Tuberculous peritonitis occurs at all ages. It is common in children associated with intestinal and mesenteric disease. The incidence is most frequent between the ages of twenty and forty. It may occur in advanced life. In one of my cases the patient was eighty-two years of age. Of 357 cases collected from the literature,* there were under ten years, 27; between ten and twenty, 75; from twenty to thirty, 87; between thirty and forty, 71; from forty to fifty, 61; from fifty to sixty, 19; from sixty to seventy, 4; above seventy, 2. In America it is more common in the negro than in the white race.

Symptoms.—In certain special features the tuberculous varies considerably from other forms of peritonitis. It presents a symptom-complex of extraordinary diversity.

In the first place, the process may be *latent* and not cause a single symptom. Such are the cases met with accidentally in the operation for

* Johns Hopkins Hospital Reports, vol. ii.

hernia or for ovarian tumor. In direct contrast are the instances in which the onset is so sudden and violent that the diagnosis of *enteritis* or *hernia* is made. The operation for strangulated hernia has, indeed, been performed. Many cases set in acutely with fever, abdominal tenderness, and the symptoms of ordinary acute peritonitis. Cases with a slow onset, abdominal tenderness, tympanites, and low continuous fever resemble *typhoid fever* very closely, and may lead to error in diagnosis.

Ascites is frequent, but the effusion is rarely large. It is sometimes hæmorrhagic. It may simulate the effusion in cirrhosis of the liver, of which disease it is to be noted that tuberculous peritonitis is often a final complication. *Tympanites* may be present in the very acute cases, when it is due to loss of tone in the intestines, owing to inflammatory infiltration; or it may occur in the old, long-standing cases when universal adhesion has taken place between the parietal and visceral layers. *Fever* is a marked symptom in the acute cases, and the temperature may reach 103° or 104°. In many instances the fever is slight. In the more chronic cases subnormal temperatures are common, and for days the temperature may not rise above 97°, and the morning temperature may be as low as 95·5°. An occasional symptom is pigmentation of the skin, which in some cases has led to the diagnosis of Addison's disease. A striking peculiarity of tuberculous peritonitis is the frequency with which either the condition simulates or is associated with *tumor*. These may be:

(*a*) *Omental*, due to puckering and rolling of this membrane until it forms an elongated firm mass, attached to the transverse colon and lying athwart the upper part of the abdomen. This cord-like structure is found also with cancerous peritonitis, but is much more common in tuberculosis. Gairdner has called special attention to this form of tumor, and in children has seen it undergo gradual resolution. A resonant percussion note may sometimes be elicited above the mass. Though usually situated in the umbilical region, the omental mass may form a prominent tumor in the right iliac region.

(*b*) *Sacculated exudation*, in which the effusion is limited and confined by adhesions between the coils, the parietal peritonæum, the mesentery, and the abdominal or pelvic organs. This encysted exudate is most common in the middle zone, and has frequently been mistaken for ovarian tumor. It may occupy the entire anterior portion of the peritonæum, or there may be a more limited saccular exudate on one side or the other. It may lie completely within the pelvis proper, associated with tuberculous disease of the Fallopian tubes.

(*c*) In rare cases the tumor formations may be due to great retraction or thickening of the *intestinal coils*. The small intestine is found shortened, the walls enormously thickened, and the entire coil may form a firm knot close against the spine, giving on examination the idea of a solid mass. Not the small intestine only, but the entire bowel from the duodenum to the rectum, has been found forming such a hard nodular tumor.

(*d*) *Mesenteric glands*, which occasionally form very large, tumor-like masses, more commonly found in children than in adults. This condition may be confined to the abdominal glands. Ascites may coexist. The condition must be distinguished from that in children, in which, with ascites or tympanites—sometimes both—there can be felt irregular nodular masses, due to large caseous formations between the intestinal coils. No doubt in a considerable number of cases of the so-called tabes mesenterica, particularly in those with enlargement and hardness of the abdomen—the condition which the French call *carreau*—there is involvement also of the peritonæum.

The *diagnosis* of these peritoneal tumors is sometimes very difficult. The omental tumor is a less frequent source of error than any other; but, as already mentioned, a similar condition may occur in cancer. The most important problem is the diagnosis of the saccular exudation from ovarian tumor. In fully one third of the recorded cases of laparotomy in tuberculous peritonitis, the diagnosis of cystic ovarian disease had been made. The most suggestive points for consideration are the history of the patient and the evidence of old tuberculous lesions. The physical condition is not of much moment, as in many instances the patients have been robust and well nourished. Irregular febrile attacks, gastro-intestinal disturbance, and pains are more common in tuberculous disease. Unless inflamed there is usually not much fever with ovarian cysts. The local signs are very deceptive, and in certain cases have conformed in every particular to those of cystic disease. The outlines in saccular exudation are rarely so well defined. The position and form may be variable, owing to alterations in the size of the coils of which in parts the walls are composed. Nodular cheesy masses may sometimes be felt at the periphery. Depression of the vaginal wall is mentioned as occurring in encysted peritonitis; but it is also found in ovarian tumor. Lastly, the condition of the Fallopian tubes, of the lungs and of the pleuræ, should be thoroughly examined. The association of salpingitis with an ill-defined anomalous mass in the abdomen should arouse suspicion, as should also involvement of the pleura, the apex of one lung, or a testis in the male.

VI. Tuberculosis of the Alimentary Canal.

(*a*) *Lips.*—Tuberculosis of the lip is very rare. It occurs occasionally in the form of an ulcer, either alone or more commonly in association with laryngeal or pulmonary disease. Two cases are reported and the literature analyzed in Verneuil's Études.* The ulcer is usually very sensitive and may be mistaken for a chancre or an epithelioma. The diagnosis may be made in cases of doubt by inoculation or the examination of a portion for tubercle bacilli.

* Tome iii, Fas. I.

(*b*) *Tongue.*—The disease begins by an aggregation of small granular bodies on the edge or dorsum. Ulceration proceeds, leaving an irregular sore with a distinct but uneven margin, and a rough, often caseous base. The disease extends slowly and may form an ulcer of considerable size. I have known it to be mistaken for epithelioma and the tongue to be excised. It is rarely met with except when other organs are involved. The glands of the angle of the jaw are not enlarged and the sore does not yield to iodide of potassium, which are points of distinction between the tuberculous and the syphilitic ulcer. In doubtful cases the inoculation test should be made, or a portion excised for microscopical examination.

(*c*) Tubercles may develop on the hard or soft *palate.* In a recent case under the care of my colleague Halsted there was a rough, irregular patch on the roof of the mouth, grayish in spots, and fissured.

(*d*) Tuberculosis of the *tonsil* has been recorded in a few cases, either in the form of the miliary granules or as caseous foci. Ulceration may occur. In the acute cases the submaxillary glands may be enlarged.

(*e*) *Pharynx.*—In extensive laryngeal tuberculosis an eruption of miliary granules on the posterior wall of the pharynx is not very uncommon. In chronic phthisis an ulcerative pharyngitis, due to extension of the disease from the epiglottis and larynx, is one of the most distressing of complications, rendering deglutition acutely painful.

(*f*) A few instances occur in literature of tuberculosis of the *œsophagus.* The condition is a pathological curiosity, except in the slight extension from the larynx, which is not infrequent.

(*g*) *Stomach.*—Many cases are reported which are doubtful. Primary disease is unknown. Marfan* was able to collect only about a dozen authentic cases. Perforation of stomach occurred six times, thrice by a tuberculous gland. In Oppolzer's case an ulcer of the colon perforated the organ. In Musser's case there was a large tuberculous ulcer three by one and a half inches in extent.

(*h*) *Intestines.*—The tubercles may be (1) primary in the mucous membrane, or more commonly (2) secondary to disease of the lungs, or in rare cases the affection may (3) pass from the peritonæum.

(1) Primary intestinal tuberculosis occurs most frequently in children, in whom it may be associated with enlargement and caseation of the mesenteric glands, or with peritonitis. It may be difficult to say at the time of the autopsy whether the primary lesion has been intestinal or peritoneal. I have already referred to Woodhead's statistics showing the remarkable frequency of infection through the bowel. In adults primary intestinal tuberculosis is rare; but now and then cases occur in which the disease sets in with irregular diarrhœa, moderate fever, and colicky pains. In a few cases hæmorrhage has been the initial symptom. Regarded at first as a chronic catarrh, it is not until the emaciation becomes marked or

* Paris Thesis, 1887.

the signs of disease appear in the lungs that the true nature is apparent. Still more deceptive are the cases in which the tuberculosis begins in the cæcum and there are symptoms of typhlitis—tenderness in the right iliac fossa, constipation, or an irregular diarrhœa and fever. These signs may gradually disappear, to recur again in a few weeks, and still further complicate the diagnosis. Perforation may occur with the formation of a pericæcal abscess, or perforation into the peritonæum may take place, or in very rare instances there is partial healing with great thickening of the walls and narrowing of the lumen.

(2) Secondary involvement of the bowels is very common in chronic pulmonary tuberculosis. The lesions are chiefly in the ileum, cæcum, and colon. The affection begins in the solitary and agminated glands or on the surface of or within the mucosa. The caseation and necrosis lead to ulceration, which may be very extensive and involve the greater portion of the mucosa of the large and small bowels. In the ileum the Peyer's patches are chiefly involved and the ulcer may be ovoid, but in the jejunum and colon the ulcers are usually round or transverse to the long axis. The tuberculous ulcer has the following characters: (*a*) It is irregular, rarely ovoid or in the long axis, more frequently girdling the bowel. (*b*) The edges and base are infiltrated, often caseous. (*c*) The submucosa and muscularis are usually involved; and (*d*) on the serosa may be seen colonies of young tubercles or a well-marked tuberculous lymphangitis. Perforation and peritonitis are not uncommon events in the secondary ulceration. Stenosis of the bowel from cicatrization may occur; the strictures may be multiple.

Tuberculosis of the *rectum* has a special interest in connection with *fistula in ano*, which, according to Spillman's statistics, occurs in about 3·5 per cent of cases of pulmonary disease. In many instances the lesion has been shown to be tuberculous. It is very rarely primary, but if the tissue on removal contains bacilli and is infective the lungs are almost invariably found to be involved. It is a common opinion that the pulmonary symptoms may develop rapidly after the fistula is cut. This may have some basis if the operation consists in laying the tract open, and not in a free excision.

(3) Extension from the peritonæum may excite tuberculous disease in the bowels. The affection may be primary in the peritonæum or extend from the tubes in women or the mesenteric glands in children. The coils of intestines become matted together, caseous and suppurating foci develop between the folds, and perforation may take place between the coils at several different places.

VII. Tuberculosis of the Liver.

This organ is very constantly involved in (*a*) general tuberculosis. The miliary granulation may be very small and in acute cases scarcely perceptible. The liver is pale and often fatty.

(*b*) A remarkable condition of the organ is produced by the development of the tubercles in the finer bile-vessels. They may attain a considerable size and are almost always softened in the centre, resembling small abscesses. The contents are always bile-stained. The organ may be honeycombed with these tuberculous abscesses.

(*c*) Large, coarse caseous masses are occasionally found, sometimes in association with perihepatitis or tuberculous peritonitis. They may attain the size of an orange or larger.

(*d*) Tuberculous cirrhosis. With the eruption of miliary tubercles there may be slight increase in the connective tissue, which is overshadowed by the fatty change. In all the chronic forms of tubercle in this organ there may be fibrous overgrowth. Hanot, who has described several varieties, states that the condition may be primary. Practically it is very rare, except in connection with chronic tuberculous peritonitis and perihepatitis, when the organ may be much deformed by a sclerosis involving the portal canals.

In this last group there may be symptoms of ascites; as a rule, tuberculosis of the liver has a purely anatomical interest.

VIII. Tuberculosis of the Brain and Cord.

Tuberculosis of the *brain* occurs as (*a*) an acute miliary infection causing meningitis and acute hydrocephalus; (*b*) as a chronic meningo-encephalitis, usually localized, and containing small nodular tubercles; and (*c*) as the so-called solitary tubercle. Between the last two forms there are all gradations, and it is rare to see the meninges uninvolved. The acute variety has already been considered. I shall here consider the chronic form, which develops slowly and has the clinical characters of a tumor.

It is most common in the young. Of 148 cases collected by Pribram 118 were under fifteen years of age. Other organs are usually involved, particularly the lungs, the bronchial glands, or the bones. In rare instances no tubercles are found elsewhere. They occur most frequently in the cerebellum; next in the cerebrum and then in the pons. The growths are often multiple, in 100 out of 183 cases (Gowers). They range in size from a pea to a walnut; larger tumors occasionally occur, and sometimes an entire lobe of the cerebellum is affected. On section the tubercle presents a grayish-yellow, caseous appearance, usually firm and hard, and encircled by a translucent, softer tissue. The centre of the growth may be semi-diffluent. As in other localities the tubercle may calcify. The tu-

mors are as a rule attached to the meninges, often to the pia at the bottom of a sulcus so that they look imbedded in the brain-substance. About the longitudinal fissure there may be an aggregation of the growths, with compression of the sinus, and the formation of a thrombus. The tuberculous tumor not infrequently excites acute meningitis. In localized meningo-encephalitis the pia is thickened, tubercles are adherent to the under surface and grow about the arteries. It is often combined with cerebral softening from interference with the circulation. Several of the most characteristic instances which I have seen were on the meninges covering the insula. This form may develop in pulmonary tuberculosis, causing hemiplegia or aphasia which may persist for months.

The symptoms of tuberculous growths in the brain are those of tumor, and will be considered in the section on the brain.

In the *spinal cord* the same forms are found. The acute tuberculous meningitis has been considered and is almost always cerebro-spinal. The solitary tubercle of the cord is rare. Herter has reported three cases and collected twenty-four instances from the literature. It was secondary in all save one case. The symptoms are those of spinal tumor or meningitis.

IX. Tuberculosis of the Genito-urinary System.

(*a*) **Tuberculosis of the Kidneys** (*Phthisis renum*).—In general tuberculosis the kidneys frequently present scattered miliary tubercles. In pulmonary tuberculosis it is common to find a few nodules in the substance of the organ, or there may be pyelitis. Primary tuberculosis of the kidneys is not very rare. In a majority of the cases the process involves the pelvis and the ureter as well, sometimes the bladder and prostate. In only one of eight cases was the prostate involved. It may be difficult to say in advanced cases whether the disease has started in the bladder, prostate, or vesicles, and crept up the ureters, or whether it started in the kidneys and proceeded downward. In a majority of cases it is, I believe, the latter, and the infection is through the blood. One kidney alone may be involved, and the disease creeps down the ureter and may only extend a few millimetres on the vesical mucosa. In a recent instance a man with aortic insufficiency, who had no lesions in the lungs, presented a localized patch in the pelvis, involving a pyramid, while the ureter, five centimetres from the bladder and at its orifice, was thickened and tuberculous. The prostate showed an area of caseation. It is most common in the middle period of life, but it may occur at the extremes of age. It is more frequent in men than in women. In the earliest stage, which may be met with accidentally, the disease is seen to begin in the pyramids and calyces. Necrosis and caseation proceed rapidly, and the colonies of tubercles start throughout the pyramids and extend upon the mucous membrane of the pelvis. As a rule, from the outset, it is a tuberculous pyo-nephrosis. The disease may be confined to one kidney, or progress more extensively in

one than in the other. At autopsy both organs are usually found enlarged. One organ may be completely destroyed and converted into a series of cysts containing cheesy substance; a form of kidney which the older writers called scrofulous. In the putty-like contents of these cysts lime salts may be deposited. In other instances the walls of the pelvis are thickened and cheesy, the pyramids eroded, and caseous nodules are scattered through the organ, even to the capsule, which may be thickened and adherent. The other organ is usually less affected, and shows only pyelitis or a superficial necrosis of one or two pyramids. The ureters are usually thickened and the mucous membrane ulcerated and caseous. Involvement of the bladder, vesiculæ seminales, and testes is not uncommon in males.

The *symptoms* are those of pyelitis. The urine may be purulent for years, and there may be little or no distress. When the bladder becomes involved micturition is frequent, and many instances are mistaken for cystitis. The condition is for many years compatible with fair health. The curability is shown by the accidental discovery of the so-called scrofulous kidney, converted into cysts containing a putty-like substance. In cases in which the disease becomes advanced and both organs are affected, constitutional symptoms are more marked. There is irregular fever, with chills, and loss of weight and strength. General tuberculosis is common. In only one of my cases were the lungs uninvolved. In a case at the Montreal General Hospital a cyst perforated and caused fatal peritonitis.

Physical examination may detect special tenderness on one side, or the kidney may be palpable in front on deep pressure; but tuberculous pyelonephritis seldom causes a large tumor. Occasionally the pelvis becomes enormously distended; but this is rare in comparison with calculous pyelitis. The urine presents changes similar to those of ordinary calculous pyelitis — pus-cells, epithelium, and occasionally definite caseous masses. Albumen is, of course, present. Tubercle bacilli may be demonstrated by the ordinary methods. Tube-casts are not often seen.

To distinguish the condition from calculous pyelitis is often difficult. Hæmorrhage may be present in both, though not nearly so frequently in the tuberculous disease. Careful examination of the pus for tubercle bacilli gives most important information. The lungs or other organs may be tuberculous.

The incidence of renal in uro-genital tuberculosis may be gathered from Orth's Göttingen material, analyzed by Oppenheim. Of 60 cases there were 34 in which the kidneys were involved.

(*b*) **Tuberculosis of the Ureters and Bladder.**—This rarely occurs as a primary affection, but is nearly always secondary to involvement of other parts, particularly the pelvis of the kidney. In the case of uro-genital tuberculosis, above mentioned, in a patient who died of heart disease, the ureter, just where it enters the bladder, showed a fresh patch of tuberculosis.

Protracted cystitis, which has come on without apparent cause, is

always suggestive of tuberculosis. The renal regions, the testes, and the prostate should be examined with care. It may follow a pyelo-nephritis or be associated with primary disease of the prostate or vesiculæ seminales.

(*c*) **Tuberculosis of the Prostate and Vesiculæ Seminales.**—The prostate is frequently involved in tuberculosis of the uro-genital tract. In Krzyincki's cases, of 15 males the prostate was involved in 14 and the vesiculæ seminales in 11. In Orth's cases the prostate was involved in 18 of the 37 cases in males. These parts are much more frequently involved than ordinary post-mortem statistics indicate.

(*d*) **Tuberculosis of the Testes.**—This somewhat common affection may be primary, or, more frequently, is secondary to tuberculous disease elsewhere. Many cases occur before the second year, and it is stated to have been met with in the fœtus. In infants it is serious and usually associated with tuberculous disease in other parts. In nine cases recently reported by Hutinel and Deschamps* in every one there was a general affection. In 20 cases reported by Jullien† 6 were under one year, and 6 between one and two years old. In five of the cases both testicles were affected. Koplik holds that most of the cases of this kind are congenital, in Baumgarten's sense. In the adult the tubercles begin within the substance of the gland, but in children the tunica albuginea is first affected. The tubercle does not always undergo caseation, but it may present a number of embryonic cells, not unlike a sarcoma.

Tubercle of the testes is most likely to be confounded with syphilis. In the latter the body of the organ is most often affected, there is less pain, and the outlines of the growth are more nodular and irregular. In obscure peritoneal disease the detection of tubercle in a testis has not infrequently led to a correct diagnosis. The association of the two conditions is not uncommon. The lesion in the testis may heal completely, or the disease may become generalized. General infection has followed operation.

(*e*) **Tuberculosis of the Fallopian Tubes, Ovaries, and Uterus.**—The special attention which has been paid to local affections of these parts by gynæcologists has taught us that primary tuberculosis of the tubes is not at all uncommon. Within a year my colleague, Kelly, has operated upon five or six cases. The disease may be primary and produce a most characteristic form of salpingitis, in which the tubes are enlarged, the walls thickened and infiltrated, and the contents cheesy. Adhesion takes place between the fimbriæ and the ovaries, or the uterus may be invaded. The condition is usually bilateral. It may occur in young children. Although, as a rule, very evident to the naked eye, there are specimens resembling ordinary salpingitis, which show on microscopical examination numerous miliary tubercles (Welch and Williams). Tuberculous salpingitis may

* Archives Générales de Médecine, 1891. † Ibid., 1890.

cause serious local disease with abscess formation, and it may be the starting-point of peritonitis.

Tuberculosis of the uterus is very rare. Only three examples have come under my observation, all in connection with pulmonary phthisis. It may be primary. The mucosa of the fundus is thickened and caseous, and tubercles may be seen in the muscular tissue. Occasionally the process extends to the vagina.

X. Arteries.

Primary tuberculosis of the larger blood-vessels is unknown. The disease may, however, occur in a large artery and not result from external invasion. In a case of chronic phthisis from my ward Councilman found a fresh tuberculous growth in the aorta, which had no connection with cheesy masses outside the vessel.

In the lungs and other organs attacked by tuberculosis the arteries are involved in an acute infiltration which usually leads to thrombosis, or tubercles may develop in the walls and proceed to caseation and softening frequently with the result of hæmorrhage. By extension into vessels, particularly veins, the bacilli are widely distributed. In meningitis tuberculosis of the arteries plays an important *rôle*.

XI. The Prognosis in Tuberculosis.

Not all persons in whose bodies the bacilli gain a foothold present marked signs of tuberculosis. As will be stated in the next section, local disease is found in a considerable number of all cadavers. Infection does not necessarily mean the establishment of a progressive and fatal disease. In my autopsies, excluding cases dead of pulmonary phthisis, 7·5 per cent presented tuberculous lesions of the lungs—a low percentage in comparison with other records, as I carefully excluded the simple fibroid puckering at the apex and the solitary cheesy nodule, unless surrounded by colonies of tubercles.

In many cases a natural or spontaneous cure is effected, for the conditions favorable to the development of the disease are not present—in other words, the tissue-soil is unsuitable. Apart from this group, a majority of which probably do not show any sign of disease, there may be spontaneous arrest after the symptoms have become decided. Many years ago Flint called attention to the self-limitation and intrinsic tendency to recovery in well-marked pulmonary tuberculosis. Of his 670 cases, 44 recovered, and in 31 the disease was arrested, spontaneously in 23 of the first group and in 15 of the second. This natural tendency to cure is still more strikingly shown in lymphatic and bone tuberculosis.

The following may be considered favorable circumstances in the prognosis of pulmonary tuberculosis: A good family history, previous good health, a strong digestion, a suitable environment, and an insidious onset,

without high fever, and without extensive pneumonic consolidation. Cases beginning with pleurisy seem to run a more protracted and more favorable course. Repeated attacks of hæmoptysis are unfavorable. When well established the course of tuberculosis in any organ is marked by intervals of weeks or months in which the fever lessens, the symptoms subside, and there is improvement in the general health.

In pulmonary cases the duration is extremely variable. Laennec placed the average duration at two years, and for the majority of cases this is perhaps a correct estimate. Pollock's large statistics of over 3,500 cases shows a mean duration of the disease of over two years and a half. Williams's analysis of 1,000 cases in private practice shows a much more protracted course, as the average duration was over seven years.

Under the subject of prognosis comes the question of the marriage of persons who have had tuberculosis, or in whose family the disease prevails. The following brief statements may be made with reference to it:

(*a*) Subjects with healed lymphatic or bone tuberculosis marry with personal impunity and may beget healthy children. It is undeniable, however, that in such families, scrofula, caries of the bone, arthritis, cerebral and pulmonary tuberculosis are more common. Which is it, "hérédité de graine ou hérédité de terrain," as the French have it, the seed or the soil, or both? We cannot yet say. The risks, however, are such as may properly be taken.

(*b*) The question of marriage of a person who has arrested or cured lung tuberculosis is more difficult to decide. If a male, the personal risk is not so great; and when the health and strength are good, the external environment favorable, and the family history not extremely bad, the experiment—for it is such—is often successful, and many healthy and happy families are begotten under these circumstances. In women the question is complicated with that of child-bearing, which increases the risks enormously. With a localized lesion, absence of hereditary taint, good physique, and favorable environment, marriage might be permitted. When tuberculosis has existed, however, in a girl whose family history is bad, whose chest expansion is slight, and whose physique is below the standard, the physician should, if possible, place his veto upon marriage.

(*c*) With existing disease, fever, bacilli, etc., marriage should be absolutely prohibited. Pregnancy and parturition hasten the process in almost every case. There is much truth, indeed, in the remark of Dubois: "If a woman threatened with phthisis marries, she may bear the first accouchement well; a second, with difficulty; a third, never."

XII. Prophylaxis in Tuberculosis.

(*a*) *General.*—The sputa of phthisical patients should be carefully collected and destroyed. Patients should be urged not to spit about carelessly, but always to use a spit-cup. Several forms of portable flasks have

been devised and are now on sale. The destruction of the sputa of consumptives should be a routine measure in both hospital and private practice. Thorough boiling or putting it into the fire is sufficient. It should be explained to the patient that the only risk, practically, is from this source. The chances of infection are greater in young children. The nursing and care of consumptives involve very slight risks indeed if proper precautions are taken. The patient should occupy a single bed.

A second important general prophylactic measure relates to the inspection of dairies and slaughter-houses. The possibility of the transmission of tuberculosis by infected milk has been fully demonstrated, and in the interest of public health the state should take measures to stamp out tuberculosis in cattle. Systematic veterinary inspection of dairies, particularly in the large cities, should be made, and full power granted to confiscate and kill suspected animals. The abattoirs should be under skilled veterinary control, and the carcasses of animals with advanced tuberculosis confiscated. There is, however, much less danger of infection through meat than through milk.

(*b*) *Individual.*—A mother with pulmonary tuberculosis should not suckle her child. An infant born of tuberculous parents, or of a family in which consumption prevails, should be brought up with the greatest care and guarded most particularly against catarrhal affections of all kinds. Special attention should be given to the throat and nose, and on the first indication of mouth-breathing, or any obstruction of the nasopharynx, a careful examination should be made for adenoid vegetations. The child should be clad in flannel and live in the open air as much as possible, avoiding close rooms. It is a good practice to sponge the throat and chest night and morning with cold water. Special attention should be paid to diet and to the mode of feeding. The meals should be at regular hours and the food plain and substantial. From the outset the child should be encouraged to drink freely of milk. Unfortunately, in these cases there seems to be an uncontrollable aversion to fats of all kinds. As the child grows older, systematically regulated exercise or a course of pulmonary gymnastics may be taken. In the choice of an occupation preference should be given to an out-of-door life. Families with a marked predisposition to tuberculosis should, if possible, reside in an equable climate. It would be best for a young man belonging to such a family to remove to Colorado or southern California, or to some other suitable climate, before trouble begins.

The trifling ailments of children should be carefully watched. In the convalescence from the fevers, which so frequently prove dangerous, the greatest caution should be exercised to prevent catching cold. Cod-liver oil, the syrup of iodide of iron, and arsenic may be given. As mentioned, care of the throat in these children is very important. When the tonsils are chronically enlarged they should be removed.

XIII. Treatment of Tuberculosis.

I. **The Natural or Spontaneous Cure.**—The spontaneous healing of local tuberculosis is an every-day affair. Many cases of adenitis and disease of the bone or of the joints terminate favorably without the aid of medicines. The healing of pulmonary tuberculosis is shown clinically by the recovery of patients in whose sputa elastic tissue and bacilli have been found; anatomically, by the presence of lesions in all stages of repair. In the granulation products and associated pneumonia a scar-tissue is formed, while the smaller caseous areas become impregnated with lime salts. To such conditions alone should the term healing be applied. When the fibroid change encapsulates but does not involve the entire tuberculous tissue, the tubercle may be termed involuted or quiescent, but is not destroyed. When cavities of any size have formed, healing, in the proper sense of the term, does not occur. I have yet to see a specimen which would indicate that a vomica had cicatrized. Cavities may be greatly reduced in size—indeed, an entire series of cavities may be so contracted by sclerosis of the tissue about them that an upper lobe, in which this process most frequently occurs, may be reduced to a third of its ordinary dimensions. Laennec understood thoroughly this natural process of cure in tuberculosis, and recognized the frequency with which old tuberculous lesions occurred in the lungs. He described *cicatrices complètes* and *cicatrices fistuleuses*, the latter being the shrunken cavities communicating with the bronchi; and suggested that, as tubercles growing in the glands, which are called scrofula, often heal, why should not the same take place in the lungs?

There is an old German axiom, "*Jedermann hat am Ende ein bischen Tuberculose*," a statement partly borne out by the statistics showing the proportion of cases in persons dying of all diseases in whom quiescent or tuberculous lesions are found in the lungs. We find at the apices the following conditions, which have been held to signify healed tuberculous processes: (1) Thickening of the pleura, usually at the posterior surface of the apex, with subadjacent induration for a distance of a few millimetres. This has, perhaps, no greater significance than the milky patch on the pericardium. (2) Puckered cicatrices at the apex, depressing the pleura, and on section showing a large pigmented, fibrous scar. The bronchioles in the neighborhood may be dilated, but there are neither tubercles nor cheesy masses. This may sometimes, but not always, indicate a healed tuberculous lesion. (3) Puckered cicatrices with cheesy or cretaceous nodules, and with scattered tubercles in the vicinity. (4) The *cicatrices fistuleuses* of Laennec, in which the fibroid puckering has reduced the size of one or more cavities which communicate directly with the bronchi.

In 1,000 autopsies, excluding the 216 cases dead of phthisis, there were 59 cases (7·5 per cent) which presented undoubted tuberculous lesions in

the lungs. I excluded the simple fibroid puckering and the solitary cheesy nodules, unless, in the latter case, there were colonies of tubercles in the vicinity. These 59 cases died of various diseases and at various ages. A majority of them were between forty and sixty. My experience tallies closely with the larger analysis made by Heitler of the Vienna post-mortem records, in which, of 16,562 cases in which the death was not directly caused by phthisis, there were 780 instances of obsolete tubercle—a percentage of 4·7. He excluded, as I have done, the simple fibroid induration. Various observations have been made of late in which the percentage ranges from twenty-seven (Bollinger) to thirty-nine (Massini). In 200 autopsies, in which this point was specially examined, Harris found 38·8 per cent in which there were relics of former active tuberculosis. The statement is made by Bouchard that, of the post-mortems at the Paris morgue—generally upon persons dying suddenly—the percentage found with some evidence of tuberculous lesion, active or obsolete, is as high as seventy-five. These figures show the extraordinary frequency of pulmonary infection and the encouraging fact that in so large a percentage the disease remains local and undergoes a process of arrest or healing.

II. **General Measures.**—There are three indications—first, to place the patient in surroundings most favorable for the maintenance of a maximum degree of nutrition; second, to take such measures as, in a local or general way, influence the tuberculous processes; third, to alleviate symptoms.

The question of environment is of first importance in the treatment of tuberculosis. It is illustrated in an interesting and practical way by an experiment of Trudeau, showing that inoculated rabbits, confined in a dark, damp place, rapidly succumb, while others, allowed to run wild, either recover or show slight lesions. It is the same in human tuberculosis. A patient confined to the house—particularly in the close, overheated, stuffy dwellings of the poor, or treated in a hospital ward—is in a position analogous to the rabbit confined to a hutch in the cellar; whereas a patient living in the fresh air and sunshine for the greater part of the day has chances comparable to those of the rabbit running wild.

In the majority of cases the treatment has to be carried out at home and often under adverse conditions. Still, much can be done if the patient is kept out of doors in the fresh air for the greater part of each day. In pulmonary tuberculosis *neither the cough, the fever, the night-sweats, nor the hæmoptysis contra-indicates this rule.* Only when the weather is blustering or rainy should the patient remain in the house. It is remarkable how quickly improvement in many instances follows this fresh-air treatment. In cities the patient can be wrapped up and placed on a sofa or in a reclining-chair on the balcony or even in the yard.

The climatic treatment of tuberculosis is simply a modification of this plan. The requirements of a suitable climate are a *pure atmosphere*, an *equable temperature* not subject to rapid variations, and a *maximum*

amount of sunshine. Given these three factors, and it makes little difference *where* a patient goes so long as he lives an outdoor life.

The *purity* of the atmosphere is the first consideration, and it is this requirement that is met so well in the mountains and forests. Altitude is a secondary consideration. The rarefaction of the air in high altitudes is of benefit in increasing the respiratory movements in pulmonary disease, but brings about in time a condition of dilatation of the air-vesicles and a permanent increase in the size of the chest which is a marked disadvantage when such persons attempt subsequently to reside at the sea-level.

The temperature of the air is also a minor consideration, so long as it is tolerably equable and not subject to rapid variations. The winter climates of the Adirondacks, of Colorado, or of Davos have the advantage of a steady cold combined with sunshine, just as the resorts of the Southern States and California, and of the south of France and Italy, have a tolerably uniform high temperature with the maximum amount of sunshine. The dryness of the air is certainly an important though not an essential factor. That it is not essential is seen in the good results obtained in the resorts at the sea-level, such as Florida, or even Torquay or Falmouth, on the south coast of England—one of the most humid atmospheres in the world.

Other considerations which should influence the choice of a locality are good accommodations and good food. Very much is said concerning the choice of locality in the different stages of pulmonary tuberculosis, but when the disease is limited to an apex, in a man of fairly good personal and family history, the chances are that he may fight a winning battle if he lives out of doors in any climate, whether high, dry, and cold or low, moist, and warm. With bilateral disease and cavity formation there is but little hope of permanent cure, and the mild or warm climates are preferable.

Whether a patient should go from home or not is a grave question which the physician is called upon to decide. It is undoubtedly, in many instances, a positive hardship to send away a patient with tolerably advanced tuberculosis. With well-marked cavities, hectic fever, night-sweats, and emaciation he is better at home, and the physician should not be too much influenced by the importunities of the patient or his friends. Advanced cases and persons with feeble hearts should never be sent to high altitudes. Of American resorts I prefer the Adirondacks for early cases. The patient should go in October, so as to become gradually accustomed to the cold. It is accessible, the winter climate is admirable, and the camp-life delightful. As the reports of Saranac Sanitarium show, recent tuberculosis does remarkably well. Personally I have seen better results from the Adirondacks than from any other place. Colorado and southern California have this advantage for early cases—they are progressive, prosperous countries in which a man may find means of livelihood and live in comfort.*

* On the question of climate, Yeo's work may be consulted with advantage.

Under this section reference may be made to the question of the treatment of tuberculosis in sanitaria. The larger cities should build special institutions within easy access by railway, with pleasant surroundings, in which early cases of pulmonary tuberculosis among the poor could be systematically treated. Advanced cases should not be admitted, but should be cared for in separate wards of the city hospitals. Sanitaria for the care of recent pulmonary tuberculosis among the well-to-do classes are also urgently needed. The results obtained at Falkenstein near Frankfurt a. M. (which certainly has nothing special, as far as climate is concerned) and at the Saranac Sanitarium illustrate how much can be done by method and care.

III. Measures which, by their Local or General Action, influence the Tuberculous Process.—Under this heading we may consider the specific, the dietetic, and the general medicinal treatment of tuberculosis.

(*a*) *The Specific Treatment.*—A glycerin extract of the cultures of tubercle bacilli was found by Koch to have a specific action upon tuberculous tissue. The influence of this tuberculin, as it is called, is best seen in lupus, upon which it exercises an extraordinary effect, unique in the history of the action of remedies. An injection of one milligramme is followed, in a few hours, by intense constitutional and local reaction. The affected tissues swell enormously, and the adjacent parts are deeply congested. Crusts form upon the surface, the swelling and inflammation gradually subside, and after several injections the lupus masses gradually disappear and are replaced by a white cicatricial tissue. Even in advanced cases of long duration the action is, in a majority of cases, prompt and beneficial. There is a great difficulty, however, in getting rid of the final remnants of the lupus tissue, and a combination of scraping with the tuberculin will probably always be needed.

In internal tuberculosis the remedy, in very early cases, may, as shown by Koch's reports, prove actually curative; unfortunately, it was employed in all classes of cases. In pulmonary tuberculosis it is a remedy to be used with the greatest caution. Of twenty-three cases in which we have used it at the Johns Hopkins Hospital, only three were benefited; in the others the action was either negative or actually detrimental. It should not be employed in cases with fever or with much consolidation. In many cases it seems to aggravate the general and local symptoms.

We are at present in the reaction wave, after being buoyed up by hopes that at last a remedy had been obtained which was positively curative in all forms of tuberculous lesions. It will probably be several years before we can speak with decision upon the true position of this remedy. Meanwhile our knowledge warrants us in urging extreme caution in its use. The recent reports of Schede indicate that the remedy has a very positive value in tuberculous arthritis when combined with other measures.

(*b*) *Dietetic Treatment.*—The outlook in tuberculosis depends much

upon the digestion. It is rare to see recovery in a case in which there is persistent gastric trouble, and the physician should ever bear in mind the fact that in this disease the *primæ viæ* control the position. The early nausea and loss of appetite in many cases of phthisis are serious obstacles. Many patients loathe food of all kinds. A change of air, or a sea voyage will promptly restore the appetite. When this is impossible, and if, as is almost always the case, fever is present, the patient should be placed at rest, kept in the open air nearly all day, and fed at stated intervals with small quantities either of milk, buttermilk, or koumyss, alternating if necessary with meat juice and egg albumen. Some cases which are disturbed by eggs and milk do well on koumyss. It may be necessary to resort to Débove's method of over-alimentation or forced feeding. The stomach is first washed out with cold water, and then, through the tube, a mixture is given containing a litre of milk, an egg, and one hundred grammes of very finely powdered meat. This is given three times a day. Sometimes the patients will take this mixture without the unpleasant necessity of the stomach-tube, in which case a smaller amount may be given. I can speak of the advantage of this plan in cases in which the gastric symptoms have been obstinate and distressing, and the general expression of opinion is, in such instances, very favorable to this plan of treatment.

In many cases the digestion is not at all disturbed and the patient can take an ordinary diet. It is remarkable how rapidly the appetite and digestion improve on the fresh-air treatment, even in cases which have to remain in the city. Care should be taken that the medicines do not disturb the stomach. Not infrequently the sweet syrups used in the cough mixtures, cod-liver oil, creasote, and the hypophosphites produce irritation, and by interfering with digestion do more harm than good. On the other hand, the bitter tonics, with acids, and the various malt preparations are often in these cases most satisfactory. The indications for alcohol in tuberculosis are enfeebled digestion with fever, a weak heart, and rapid pulse. A routine administration is not advisable, and there is no evidence that its persistent use promotes fibroid processes in the tuberculous areas. In the advanced stages, particularly when the temperature is low between eight and ten in the morning, whisky and milk, or whisky, egg, and milk may be given with great advantage. The red wines are also beneficial in moderate quantities.

(*c*) *General Medical Treatment.*—No medicinal agents have any special or peculiar action upon tuberculous processes. The influence which they exert is upon the general nutrition, increasing the physiological resistance and rendering the tissues less susceptible to invasion. The following are the most important remedies which seem to act in this manner:

Creasote, which may be administered in capsules, in increasing doses, beginning with one minim three times a day and, if well borne, increasing the dose to eight or ten minims. It may also be given in solution

with tincture of cardamom and alcohol. It is an old remedy, strongly recommended by Addison, and the reports of Jaccoud, Fraentzel, and many others show that it has a positive value in the disease. Guaiacol may be given as a substitute, either internally or hypodermically. In 101 cases in which it was used at my clinic, by Meredith Reese, the chief action was on the cough and expectoration, which were much lessened, but the remedy had no essential influence on the progress of the disease.

Cod-liver Oil.—In glandular and bone tuberculosis, this remedy is undoubtedly beneficial in improving the nutrition. In pulmonary tuberculosis its action is less certain, and it is scarcely worthy of the unbounded confidence which it enjoyed for so many years. It should be given in small doses, not more than a teaspoonful three times a day after meals. It seems to act better in children than in adults. When it is not well borne, a dessertspoonful of rich cream three times a day is an excellent substitute. The clotted or Devonshire cream is preferable.

The Hypophosphites.—These in various forms are useful tonics, but it is doubtful if they have any other action. They certainly exercise no specific influence upon tubercle. They may be given in the form of the syrup of the hypophosphites of calcium, sodium, and potassium of the U. S. P.

Arsenic.—There is no general tonic more satisfactory in cases of tuberculosis of all kinds than Fowler's solution. It may be given in five-minim doses three times a day and gradually increased; stopping its use whenever unpleasant symptoms arise, and in any case intermitting it every third or fourth week.

One or two special methods of dealing with pulmonary tuberculosis may here be mentioned. The local treatment, by direct injection into the lungs, has been practised since its strong advocacy by Pepper. It has, however, not gained the general support of the profession, and is occasionally followed by serious results. As a rule, it may be practised with impunity, and the injections may be made with a long hypodermic needle into any portion of the lung which is diseased. Iodine, carbolic acid, creasote (three per cent solution in almond oil), and iodoform have been used for the purpose. The remarkable results which surgeons have recently obtained in the treatment of joint tuberculosis by injections of iodoform point to this as a remedy which will probably prove of service when injected directly into the lungs.

Treatment by compressed air is in many cases beneficial, and under its use the appetite improves, there is gain in weight, and reduction of the fever. The air may be saturated with creasote.

IV. **Treatment of Special Symptoms in Pulmonary Tuberculosis.**—(*a*) *The Fever.*—There is no more difficult problem in practical therapeutics than the treatment of the pyrexia of tuberculosis. The patient should be at rest, and when practicable wheeled into the fresh air for as long a time as possible during the day. Fever does not contra-indicate an out-of-door

life, but it is well for patients with a temperature above 101° or 102° to be at rest. For the continuous pyrexia or the remittent type of the early stages, quinine, small doses of digitalis, and the salicylates may be tried; but they are uncertain and rarely reliable. Under no circumstances is that priceless remedy, quinine, so much abused as in the fever of tuberculosis. In large doses it has a moderate antipyretic action, but it is just in these efficient doses that it is so apt to disturb the stomach.

Antipyrin and antifebrin may be used cautiously; but it is better, when the fever rises above 103°, to rely upon cold sponging or the tepid bath, gradually cooled. When softening has taken place and the fever assumes the characteristic septic type, the problem becomes still more difficult. As shown by Chart XIII (which is not by any means an exceptional one), the pyrexia, at this stage, lasts only for twelve or fifteen hours. As a rule it is not more than from eight to ten hours in which the fever is high enough to demand antipyretic treatment. Sometimes antifebrin, given in two-grain doses every hour for three or four hours before the rise in temperature takes place, either prevents entirely or limits the paroxysm. If the temperature begins to rise between two and three in the afternoon, the antifebrin may be given at eleven, twelve, one, and, if necessary, at two. It answers better in this way than given in the single doses. Careful sponging of the extremities for from half an hour to an hour during the height of the fever is useful. Quinine is of little benefit in this type of fever; the salicylates still less.

(*b*) *Sweating.*—The atropine, in doses of gr. $\frac{1}{120}$–$\frac{1}{60}$, and the aromatic sulphuric acid in large doses are the best remedies. When there are cough and nocturnal restlessness, an eighth of a grain of morphia may be given with the atropine. Muscarin (♏ v of a one per cent solution), tincture of nux vomica (♏ xxx), picrotoxin (gr. $\frac{1}{60}$) may be tried. The patient should use light flannel night-dresses, as the cotton night-shirts, when soaked with perspiration, have a very unpleasant cold, clammy feeling.

(*c*) The *cough* is a troublesome, though necessary, feature in pulmonary tuberculosis. Unless very worrying and disturbing sleep at night, or so severe as to produce vomiting, it is not well to attempt to restrict it. When irritative and bronchial in character, inhalations are useful, particularly the tincture of benzoin or preparations of tar, creasote, or turpentine. The throat should be carefully examined, as some of the most irritable and distressing forms of cough in phthisis result from laryngeal erosions. The distressing nocturnal cough, which begins just as the patient gets into bed and is preparing to fall asleep, requires, as a rule, preparations of opium. Codeia, in quarter or half grain doses, or the syrupus codeiæ (℥ j) may be given. An excellent combination for the nocturnal cough of phthisis is morphia (gr. $\frac{1}{8}$–$\frac{1}{4}$), dilute hydrocyanic acid (♏ ij–iij), and syrup of wild cherry (℥ j). The spirits of chloroform, B. P., or the mistura chloroformi, U. S. P., or Hoffman's anodyne, given

in whisky before going to sleep, are efficacious. Mild counter-irritation, or the application of a hot poultice, will sometimes promptly relieve the cough. In the later stages of the disease, when cavities have formed, the accumulated secretion must be expectorated and the paroxysms of coughing are now most exhausting. The sedatives, such as morphia and hydrocyanic acid, should be given cautiously. The aromatic spirits of ammonia in full doses help to allay the paroxysm. When the expectoration is profuse, creasote internally, or inhalations of turpentine and iodine, are useful.

(*d*) For the *diarrhœa* large doses of bismuth, combined with Dover powder, and small starch enemata, with or without opium, may be given. The acetate of lead and opium pill often acts promptly, and the acid diarrhœa mixture, dilute acetic acid (♏ x–xv), morphia (gr. $\frac{1}{8}$), and acetate of lead (gr. j–ij), may be tried.

(*e*) The treatment of the hæmoptysis will be considered in the section on hæmorrhage from the lungs. Dyspnœa is rarely a prominent symptom except in the advanced stages, when it may be very troublesome and distressing. Ammonia and morphia, cautiously administered, may be used.

If the pleuritic pains are severe, the side may be strapped or painted with tincture of iodine. The dyspeptic symptoms require careful treatment, as the outlook in individual cases depends much upon the condition of the stomach. Small doses of calomel and soda often allay the distressing nausea of the early stage.

XXVII. LEPROSY.

Definition.—A chronic infectious disease caused by the *bacillus lepræ*, characterized by the presence of tubercular nodules in the skin and mucous membranes (tubercular leprosy) or by changes in the nerves (anæsthetic leprosy). At first these forms may be separate, but ultimately both are combined, and in the characteristic tubercular form there are disturbances of sensation.

Etiology.—The disease is very widely spread, and within the past few years renewed attention has been directed to it, owing to a belief that it is greatly on the increase. It is one of the oldest of known diseases. At present it prevails widely, particularly in hot countries. In India it is estimated that there are over 250,000 lepers. In Europe, where it prevailed in the middle ages, it has become almost unknown except in Norway and in the Orient. On this continent leprosy exists in the Gulf States and extensively in Mexico. At Key West Berger states that there are one hundred cases, and Blanc found forty lepers in New Orleans. A few isolated cases arrive from time to time in the cities of the Atlantic coast. In the Northwestern States a few cases exist among the Norwegian and Icelandic settlers. On the Pacific coast cases are seen not infrequently among the Chinese. An endemic focus is at Tracadie,

New Brunswick. A few cases are also met with in Cape Breton, N. S. At Tracadie, which is on a bay of the Gulf of St. Lawrence, the disease is limited to two or three counties which are settled by French Canadians. The disease was imported from Normandy about the end of the last century. The cases are confined in a lazaretto, to which they are sent so soon as the disease is manifest. I made a visit to the settlement two years ago with the medical officer, A. A. Smith, of Chatham, at which time there were only eighteen patients in the hospital. It is interesting to note that the disease has gradually diminished by segregation; formerly there were over forty under surveillance.

In the Sandwich Islands leprosy has developed to an enormous extent. Morrow states that in 1889 there were 1,100 lepers in the settlement at Molokai.

In the West Indies the disease has been long endemic, and Beavan Rake, of Trinidad, has contributed some of the most interesting of recent clinical and pathological studies.

The disease attacks all classes and persons of all ages. It is probably communicated by contagion. Inoculation was successfully performed by Arning in a Hawaiian convict. Graham, who some years ago carefully investigated the Tracadie settlement, came to the conclusion that the disease was there probably transmitted by contagion; and A. A. Smith, the present medical officer, tells me that he knows of no facts which are opposed to that view. It is, however, only contagious in the same sense as syphilis, and just as accidental contamination with this virus is extremely rare so it is with leprosy. The closest possible contact may take place for years, as between parent and child, without transmission, and not one of the Sisters of Charity who have for more than forty years so faithfully nursed the lepers at Tracadie has contracted the disease. It is difficult to explain the rapid spread of the disease in the Sandwich Islands on any other view than contagion, and yet it is strange that there is no evidence of a primary lesion or external sore comparable to that of syphilis. Morrow states that "in the immense majority of cases the disease is propagated by sexual congress."

The disappearance of the disease in the middle ages no doubt resulted directly from the isolation enforced at that time. The disease has possibly in some instances been transmitted by vaccination. Hereditary transmission cannot be excluded, and there is no good reason why the disease should not be communicated, as is syphilis, from parent to child.

Jonathan Hutchinson believes that the disease is always associated with some special kind of food, particularly fish. Though he does not deny the specific nature of the disease or the possibility of contagion, he would make apparently the fish diet the *tertium quid* which renders the patient susceptible, or, if I gather aright from his recent communication, with which the poison may be taken. The facts which are manifest at the Tracadie settlement are very much opposed to this view. If a fish diet

could alone in any way induce the disease, by this time leprosy would be wide-spread in the counties along the Gulf of St. Lawrence, as fish is the main article of diet winter and summer. There is not the slightest difference in race, the mode of life, or in the surroundings of the inhabitants in the regions adjacent to Caraquet and Tracadie, and yet leprosy has been for nearly a century limited to two or three counties.

The Bacillus Lepræ.—Hansen, of Bergen, first discovered this organism, which has many points of resemblance to the *bacillus tuberculosis*, but can be differentiated from it. It occurs in extraordinary numbers in the tuberculous tissue. It has been cultivated successfully (Babes), but inoculation experiments on animals have been negative.

Morbid Anatomy.—The leprosy tubercles consist of granulomatous tissue made up of cells of various sizes in a connective-tissue matrix. The bacilli in extraordinary numbers lie partly between and partly in the cells. The growth gradually involves the skin, producing tuberous outgrowths with intervening areas of ulceration or cicatrization, which in the face may gradually produce the so-called *facies leontina*. The mucous membranes, particularly the conjunctiva, the cornea, the larynx, may be gradually involved. In many cases deep ulcers form which result in extensive loss of substance or loss of fingers or toes, the so-called *lepra mutilans*. In anæsthetic leprosy there is a peripheral neuritis due to the development of the bacilli in the nerve-fibres. Indeed, this involvement of the nerves plays a primary part in the etiology of many of the important features, particularly the trophic changes in the skin and the disturbances of sensation.

Clinical Forms.—(*a*) **Tubercular Leprosy.**—Prior to the appearance of the nodules there are areas of cutaneous erythema which may be sharply defined and often hyperæsthetic. This is sometimes known as *macular* leprosy. The affected spots in time become pigmented. In some instances this superficial change continues without the development of nodules, the areas become anæsthetic, the pigment gradually disappears, and the skin gets perfectly white—the *lepra alba*. Among the patients at Tracadie it was particularly interesting to see three or four in this early stage presenting on the face and forearms a patchy erythema with slight swelling of the skin. The diagnosis of the condition is perfectly clear, though it may be a long time before any other than sensory changes develop. The eyelashes and eyebrows and the hairs on the face fall out. The mucous membranes finally become involved, particularly the mouth, throat, and larynx; the voice becomes harsh and finally aphonic. Death results not infrequently from the laryngeal complications and aspiration pneumonia. The conjunctivæ are frequently attacked, and the sight is lost by a leprous keratitis.

(*b*) **Anæsthetic Leprosy.**—This remarkable form has, in characteristic cases, no external resemblance whatever to the other variety. It usually begins with pains in the limbs and areas of hyperæsthesia or of numbness.

Very early there may be trophic changes, seen in the formation of small bullæ (Hillis). Maculæ appear upon the trunk and extremities, and after persisting for a variable time gradually disappear, leaving areas of anæsthesia, but the loss of sensation may come on independently of the outbreak of maculæ. The nerve-trunks, where superficial, may be felt to be large and nodular. The trophic disturbances are usually marked. Pemphigus-like bullæ develop in the affected areas, which break and leave ulcers which may be very destructive. The fingers and toes are liable to contractures and to necrosis, so that in chronic cases the phalanges are lost. The course of anæsthetic leprosy is extraordinarily chronic and may persist for years without leading to much deformity. One of the most prominent clergymen on this continent has had anæsthetic leprosy for more than thirty years, which until recently has not seriously interfered with his usefulness, and not in the slightest with his career.

Diagnosis.—Even in the early stage the dusky erythematous maculæ with hyperæsthesia or areas of anæsthesia are very characteristic. In an advanced grade neither the tubercular nor anæsthetic forms could possibly be mistaken for any other affection.

Treatment.—There are no specific remedies in the disease, and general tonics combined with local treatment meet the only available indications. The gurjun and chaulmoogra oils have been recommended, the former in doses of from five to ten minims, the latter in two-drachm doses. The cases should be isolated, although the risk of catching the disease by direct contagion is extremely slight.

XXVIII. GLANDERS (*Farcy*).

Definition.—An infectious disease of the horse, communicated occasionally to man. In the horse it is characterized by the formation of nodules, chiefly in the nares (glanders) and beneath the skin (farcy).

Etiology.—The disease belongs to the infective granulomata. The local manifestations in the nostrils and the skin of the horse are due to one and the same cause. The specific germ was discovered by Loeffler and Schütz. It is a short, non-motile bacillus, not unlike that of tubercle. It grows readily on the ordinary culture media. For the full recognition of glanders in man we are indebted to the labors of Rayer, whose monograph remains one of the best descriptions ever given of the disease. Man becomes infected by contact with diseased animals, and usually by inoculation on an abraded surface of the skin. The contagion may also be received on the mucous membrane. In one of the Montreal cases a gentleman was probably infected by the material expelled from the nostrils of his horse, which was not suspected to have the disease.

Morbid Anatomy.—As in the horse, the disease may be localized in the nose (glanders), or beneath the skin (farcy). The essential lesion

is the granulomatous tumor, characterized by the presence of numerous lymphoid and epithelioid cells, among and in which are seen the glanders bacilli. These nodular masses tend to break down rapidly, and on the mucous membrane form ulcers, while beneath the skin they form abscesses. The glanders nodules may also occur in the internal organs.

Symptoms.—An acute and a chronic form of glanders may be recognized in man, and an acute and a chronic form of farcy.

Acute Glanders.—The period of incubation is rarely more than three or four days. There are signs of general febrile disturbance. At the place of infection there are swelling, redness, and lymphangitis. Within two or three days there is involvement of the mucous membrane of the nose, the nodules break down rapidly to ulcers, and there is a mucopurulent discharge. An eruption of papules, which rapidly become pustules, breaks out over the face and about the joints. It has been mistaken for variola. This was carefully studied by Rayer and is figured in his monograph. In a Montreal case this copious eruption led the attending physician to suspect small-pox, and the patient was isolated. There is great swelling of the nose. The ulceration may go on to necrosis, in which case the discharge is very offensive. The lymph-glands of the neck are usually much enlarged. Subacute pneumonia is very apt to develop. This form runs its course in about eight or ten days, and is invariably fatal.

Chronic glanders is rare and difficult to diagnose, as it is usually mistaken for a chronic coryza. There are ulcers in the nose, and often laryngeal symptoms. It may last for months, or even longer, and recovery sometimes takes place. The diagnosis may be extremely difficult. In such cases cultures should be made and portions of the pure culture inoculated in the guinea-pig. The animal dies within thirty hours, and the testicles are found to be enormously swollen and already in the condition of abscess.

Acute farcy in man results usually from the inoculation of the virus into the skin. There is an intense local reaction with a phlegmonous inflammation. The lymphatics are early affected, and along their course there are nodular subcutaneous enlargements, the so-called farcy buds, which may rapidly go on to suppuration. There are pains and swelling in the joints and abscesses may form in the muscles. The symptoms are those of an acute infection, almost like an acute septicæmia. The nose is not involved and the superficial skin eruption is not common.

The disease is fatal in a large proportion of the cases, usually in from twelve to fifteen days.

Chronic farcy is characterized by the presence of localized tumors, usually in the extremities. These tumors break down into abscesses, and sometimes form deep ulcers, without much inflammatory reaction and without special involvement of the lymphatics. The disease may last for months or even years. Death may result from pyæmia, or occasionally

acute glanders develops. The celebrated French veterinarian, Bouley, had it and recovered.

The disease is transmissible also from man to man. Washer-women have been infected from the clothes of a patient. In the diagnosis of this affection the occupation is very important. Nowadays, in cases of doubt, the inoculation should be made in animals, as in this way the disease can be readily determined.

Treatment.—If seen early the wound should be either cut out or thoroughly destroyed by caustics, and an antiseptic dressing applied. The farcy buds should be early opened. In the acute cases there is very little hope. In the chronic cases recovery is possible, though often tedious.

XXIX. ACTINOMYCOSIS.

Definition.—A chronic inflammatory affection produced by the actinomyces or ray-fungus.

Etiology.—The disease is wide-spread among cattle, and occurs also in the pig. It was first described by Bollinger in the ox, in which it forms the affection known in this country as "big-jaw." Examples of the disease were common in the cattle killed at the abattoir in Montreal. In man the disease was first described by James Israel, and subsequently Ponfick insisted upon the identity of the disease in man and cattle.

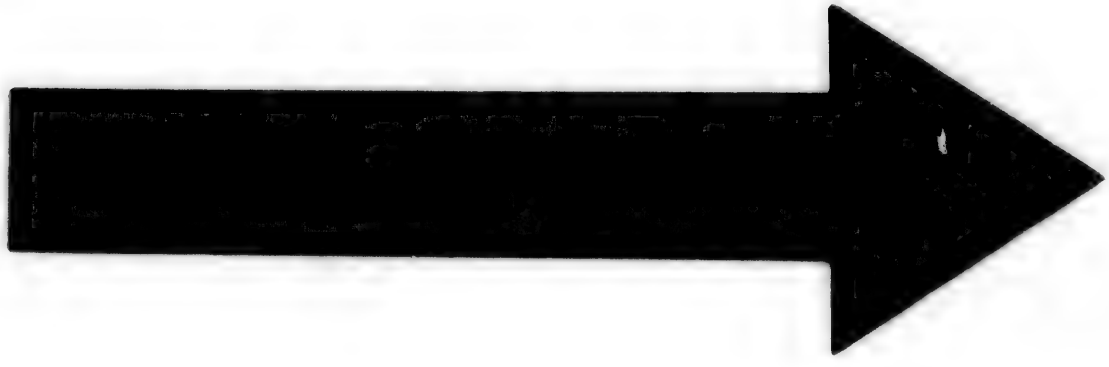
In this country and in England the disease is rare, and only a few cases have been described. Although familiar with the affection in cattle since 1878, and constantly on the lookout for the disease, no instance has fallen under my personal observation.

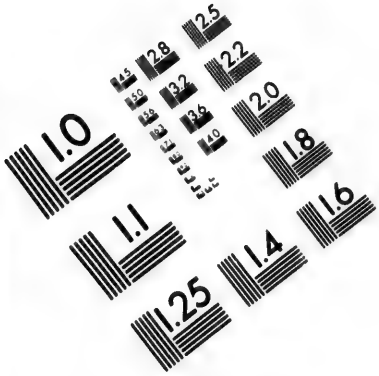
The *parasite* is a fungus belonging to the species *Cladothrix*. In both man and cattle it can be seen in the pus from the affected region as small yellowish granules from one half to two millimetres in diameter. Microscopically these bodies are seen to be made up of threads which radiate from a centre and present bulbous, club-like terminations. Böstrom has recently published an elaborate research on their structure and development.

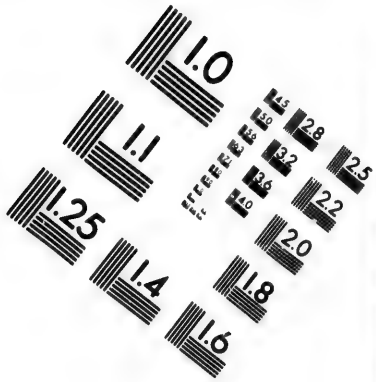
The parasite has been successfully cultivated and the disease has been inoculated, both with the natural and artificially grown fungus.

The Mode of Infection.—The fungus has not been detected outside the body. It seems highly probable that it is taken in with the food. The site of infection in a majority of cases in man and animals is in the mouth or neighboring passages. In the cow, possibly also in man, ears of barley or rye have been carriers of the fungus.

Morbid Anatomy.—In the earliest stages of its growth the parasite gives rise to a small granulation tumor, not unlike that produced by the *bacillus tuberculosis*, which contains, in addition to small round cells, epithelioid elements and giant cells. After it reaches a certain size there







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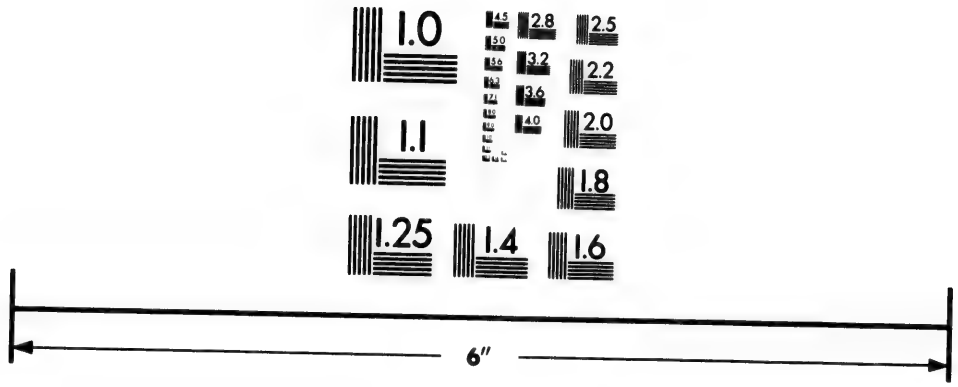

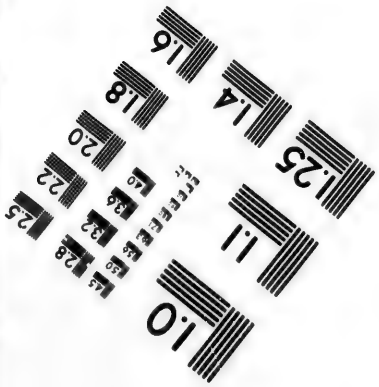

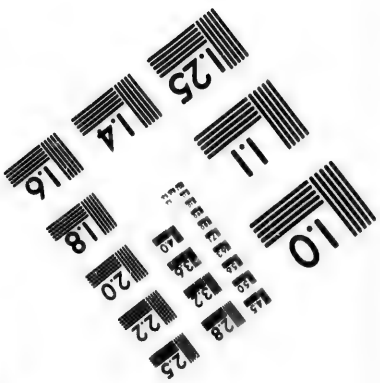

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is great proliferation of the surrounding connective tissue, and the growth may, particularly in the jaw, look like, and was long mistaken for, osteosarcoma. Finally suppuration occurs, which, according to Israel, may be produced directly by the fungus itself.

Clinical Forms.—(*a*) **Alimentary Canal.**—Israel is said to have found the fungus in the cavities of carious teeth. The jaw has been involved in a number of cases in man. The patient comes under observation with swelling of one side of the face, or with a chronic enlargement of the jaw which may simulate sarcoma. In the case described by Bodamer at the German Hospital, Philadelphia, the swelling involved the right side of the face, the temporal region, and the neck; there were numerous sinuses, and the case had the appearance of chronic necrosis of the bones.

The tongue has been involved in several cases, forming small growths, which in one instance were primary, in the others secondary to disease of the jaw. In the intestines the disease may occur either as a primary or secondary affection. At the Charité in Berlin in 1884 I saw with Oscar Israel a remarkable instance in which there were actinomycotic ulcers in the small intestines. Cases have been reported of pericæcal abscess due to the fungus. An instance of primary actinomycosis of the large intestine with metastases has also been described. The liver may be affected primarily, as in the case reported by Sharkey and Acland.

(*b*) **Pulmonary Actinomycosis.**—In September, 1878, James Israel described a remarkable mycotic disease of the lungs, which subsequent observation showed to be the affection described the year before by Bollinger in cattle. Since that date thirty-four instances have been reported in which the lungs were affected. Hodenpyl has analyzed these and reports two cases from the Roosevelt Hospital.

It is a chronic infectious disorder of the lungs, characterized by cough, fever, wasting, and a muco-purulent, sometimes fœtid, expectoration. The lesions are unilateral in a majority of the cases. Hodenpyl classifies them in three groups: (1) Lesions of chronic bronchitis; in one case the diagnosis was made by the presence of the actinomyces in the sputum. (2) Miliary actinomycosis, closely resembling miliary tubercle, but the nodules are seen to be made up of groups of fungi, surrounded by granulation tissue. This form of pulmonary actinomycosis is not infrequent in oxen with advanced disease of the jaw or adjacent structures. (3) The cases in which there is more extensive destructive disease of the lungs, bronchopneumonia, interstitial changes, and abscesses, the latter forming cavities large enough to be diagnosed during life. Actinomycotic lesions of other organs are often present in connection with the pulmonary disease: erosion of the vertebræ, necrosis of the ribs and sternum, subcutaneous abscesses, and occasionally metastases in all parts of the body.

Symptoms.—The fever is of an irregular type and depends largely on the existence of suppuration. The cough is an important symptom, and

the diagnosis in eighteen of the cases was made during life by the discovery of the actinomyces. Death results usually with septic symptoms. Occasionally there is a condition simulating typhoid fever. The average duration of the disease was ten months. Of the thirty-four cases all died except two. Clinically the disease closely resembles certain forms of pulmonary tuberculosis and of fœtid bronchitis. It is not to be forgotten in the examination of the sputum that, as Bizzozero mentions, certain degenerated epithelial cells may resemble the fungus. The radiating leptothrix threads about the epithelium of the mouth sometimes present a striking resemblance.

(*c*) **Cutaneous Actinomycosis.**—In several instances in connection with chronic ulcerative disease of the skin the ray-fungus has been found. It is a very chronic affection associated with the development of tumors which suppurate and leave open sores which may remain for years. It resembles tuberculosis of the skin.

(*d*) **Cerebral Actinomycosis.**—Bollinger has reported an instance of primary disease of the brain. The symptoms were those of tumor. A second remarkable case has been reported by Gamgee and Delepine. The patient was admitted to St. George's Hospital with left-sided pleural effusion. At the post-mortem three pints of purulent fluid were found in the left pleura; there was an actinomycotic abscess of the liver, and in the brain there were abscesses in the frontal, parietal, and temporo-sphenoidal lobes which contained the mycelium, but no clubs. A third case, reported by O. B. Keller, had *empyema necessitatis*, which was opened and actinomyces were found in the pus. Subsequently she had Jacksonian epilepsy, for which she was trephined twice and abscesses opened, which contained actinomyces grains. Death occurred after the second operation.

Diagnosis.—The disease is often mistaken for and is in reality a chronic pyæmia. The only test is the presence of the actinomyces in the pus. Metastases may occur as in pyæmia and in tumors. The tendency, however, is rather to produce a local purulent affection which erodes the bones and is very destructive. In cattle the disease may cause metastases without any suppuration; thus in a Montreal case the jaw and tongue were the seat of the most extensive disease with very slight suppuration, while the lungs presented numbers of secondary growths containing the fungus.

Treatment.—This is largely surgical and is practically that of pyæmia. Incision of the abscess, removal of the dead bone, and thorough irrigation are appropriate measures.

XXX. INFECTIOUS DISEASES OF DOUBTFUL NATURE.

(1) FEBRICULA—EPHEMERAL FEVER.

Definition.—Fever of slight duration, probably depending upon a variety of causes.

A febrile paroxysm lasting for twenty-four hours and disappearing completely is spoken of as ephemeral fever. If it persists for three, four, or more days without local affection it is referred to as febricula.

The cases may be divided into several groups:

(*a*) Those which represent mild or abortive types of the infectious diseases. It is not very infrequent, during an epidemic of typhoid, scarlet fever, or measles, to see cases with some of the prodromal symptoms and slight fever which persist for two or three days without any distinctive features. I have already spoken of these in connection with the abortive type of typhoid fever. Possibly, as Kahler suggests, some of the cases of transient fever are due to the rheumatic poison.

(*b*) In a larger and perhaps more important group of cases the symptoms develop with dyspepsia. In children indigestion and gastro-intestinal catarrh are often accompanied by fever. Possibly some instances of longer duration may be due to the absorption of certain toxic substances. Slight fever has been known to follow the eating of decomposing substances or the drinking of stale beer; but the gastric juice has remarkable antiseptic properties, and the frequency with which persons take from choice articles which are "high," shows that poisoning is not likely to occur unless there is existing gastro-intestinal disturbance.

(*c*) Cases which follow exposure to foul odors or sewer-gas. That a febrile paroxysm may follow a prolonged exposure to noxious odors has long been recognized. The cases which have been described under this heading are of two kinds: an acute severe form with nausea, vomiting, colic, and fever, followed perhaps by a condition of collapse or coma; secondly, a form of low fever with or without chills. A good deal of doubt still exists in the minds of the profession about these cases of so-called sewer-gas poisoning. It is a notorious fact that workers in sewers are remarkably free from disease, and in many of the cases which have been reported the illness may have been only a coincidence. There are instances in which persons have been taken ill with vomiting and slight fever after exposure to the odor of a very offensive post-mortem. Whether true or not, the idea is firmly implanted in the minds of the laity that very powerful odors from decomposing matters may produce sickness.

(*d*) Many cases doubtless depend upon slight unrecognized lesions, such as tonsillitis or occasionally an abortive or larval pneumonia. Children are much more frequently affected than adults.

The *symptoms* set in, as a rule, abruptly, though in some instances there may have been preliminary *malaise* and indisposition. Headache,

loss of appetite, and furred tongue are present. The urine is scanty and high-colored, the fever ranges from 101° to 103°, sometimes in children it rises higher. The cheeks may be flushed and the patient has the outward manifestations of fever. In children there may be bronchial catarrh with slight cough. Herpes on the lips is a common symptom. Occasionally in children the cerebral symptoms are marked at the outset, and there may be irritation, restlessness, and nocturnal delirium. The fever terminates abruptly by crisis from the second to the fourth day; in some instances it may continue for a week.

The *diagnosis* generally rests upon the absence of local manifestations, particularly the characteristic skin rashes of the eruptive fevers, and most important of all the rapid disappearance of the pyrexia. The cases most read. recognized are those with acute gastro-intestinal disturbance.

The *treatment* is that of mild pyrexia—rest in bed, a laxative, and a fever mixture containing nitrate of potash and sweet spirits of nitre.

(2) WEIL'S DISEASE.

Acute Febrile Icterus.—In 1886 Weil described an acute infectious disease, characterized by fever and jaundice. Much discussion has taken place concerning the true nature of this affection, but it has not been definitely determined whether it is a specific disease or only a jaundice which may be due to various causes. The majority of the cases have occurred during the summer months. The cases have occurred in groups in different cities. A few cases have been reported in this country (Lanphear). Males are most frequently affected. Many of the cases have been in butchers. The age of the patients has been from twenty-five to forty.

The disease sets in abruptly, usually without prodromata and often with a chill. There are headache, pains in the back, and sometimes intense pains in the legs and muscles. The fever is characterized by marked remissions. Jaundice appears early. The liver and spleen are usually swollen; the former may be tender. The jaundice may be light, but in many of the cases described it has been of the obstructive form, and the stools have been clay-colored. Gastro-intestinal symptoms are rarely present. The fever lasts from ten to fourteen days; sometimes there are slight recurrences, but a definite relapse is rare.

Albumen is usually present in the urine; hæmaturia has occurred in some cases.

Cerebral symptoms, delirium and coma, have been met.

In the few post-mortems which have been made nothing distinctive has been found. Its occurrence as an independent malady, apart from other infectious processes, has scarcely yet been definitely established.

(3) MILK-SICKNESS.

This remarkable disease prevails in certain districts of the United States, west of the Alleghany Mountains, and is connected with the affection in cattle known as the *trembles*. It prevailed extensively in the early settlements in certain of the Western States and proved very fatal. The general opinion is that it is communicated to man only by eating the flesh or drinking the milk of diseased animals. The butter and cheese are also poisonous. In animals, cattle and the young of horses and sheep are most susceptible. It is stated that cows giving milk do not themselves show marked symptoms unless driven rapidly, and, according to Graff, the secretion may be infective when the disease is latent. When a cow is very ill, food is refused, the eyes are injected, the animal staggers, the entire muscular system trembles, and death occurs in convulsions, sometimes with great suddenness. Nothing definite is known as to the cause of the disease. It is most frequent in new settlements.

In man the symptoms are those of a more or less acute intoxication. After a few days of uneasiness and distress the patient is seized with pains in the stomach, nausea and vomiting, fever and intense thirst. There is usually obstinate constipation. The tongue is swollen and tremulous, the breath is extremely foul and, according to Graff, is as characteristic of the disease as the odor is of small-pox. Cerebral symptoms—restlessness, irritability, coma, and convulsions—are sometimes marked, and there may gradually be produced a typhoid state in which the patient dies.

The duration of the disease is variable. In the most acute forms death occurs within two or three days. It may last for ten days, or even for three or four weeks. Graff states that insanity occurred in one case. The poisonous nature of the flesh and of the milk has been demonstrated experimentally. An ounce of butter or cheese, or four ounces of the beef, raw or boiled, three times a day will kill a dog within six days. No definite pathological lesions are known. Fortunately, the disease has become rare, and the observation of Drake, Yandell, and others, that the disease gradually disappears with the clearing of the forests and improved tillage, has been amply substantiated. It still prevails in parts of North Carolina.

(4) MALTA FEVER.

This disease, also known as Mediterranean fever, Neapolitan fever, and rock fever, has been studied particularly by the naval and military medical officers who have been stationed on the island of Malta. It prevails also in Naples and other districts of the Mediterranean. While endemic in the island of Malta, the disease in some years reaches epidemic proportions. Young persons are, as a rule, affected. The incubation may be from six to ten days.

The *symptoms* are thus briefly and clearly described in an editorial in

the British Medical Journal: "The disease declares itself gradually, with headache, sleeplessness, loss of appetite, and thirst, often without shivering or diarrhœa, and without spots. Symptoms of this kind, with more or less severity, last for three or four weeks; apparent but deceptive convalescence then usually sets in, to be followed in a few days by a relapse, with rigors, intense headache and fever, with, frequently, diarrhœa. In this state the patient may continue for five or six weeks, with more or less delirium. Improvement again sets in, to be followed, it may be, by another relapse in about ten days or a fortnight, with shivering, headache, sleeplessness, great debility, with night-sweats, pains in the hips, knees, ankles, and elbows, and often in one or both testicles. Again, the patient enters on a state of convalescence, which may last for a month or six weeks. The old symptoms may again appear, with extreme debility, a thickly coated tongue, with thirst, a temperature ranging from 105° Fahr. in the evening to nearly normal in the morning, with night-sweats bringing no relief to the general distress. The rheumatic symptoms are the most constant and the most distressing; all the joints, large and small, may suffer. Dr. Veale described cases in which the intervertebral joints, especially those of the lumbar region and the sacro-iliac synchondroses, were so severely affected that the patient "dreads every movement"; he will lie for days in one position, risking the formation of bed-sores, and resisting the desire to evacuate his bowels rather than encounter the suffering that a movement will entail. Oftentimes the tendo Achillis and the fibrous structures around the ankle-joint are involved; but perhaps the lumbar aponeuroses and the sheaths of the nerves issuing from the sacral plexus are still more commonly affected."*

The nature of the disease is still under discussion. McLean, of the Army Medical School, in 1879, suggested that it was a typho-malarial fever, and Veale called it *febris complicata*. Others have supposed that it is an anomalous form of malaria, but it does not behave like any ordinary form of paludal fever and resists quinine. This is a question which could be determined positively by the blood examination. According to Bruce, no characteristic typhoid lesions are found in fatal cases. This author has described the presence of a micrococcus in the spleen. The Italian observers have noted enlargement of the mesenteric glands, and Cantani regards it as an adeno-typhoid. The identity of Malta and the so-called rock fever of Gibraltar is, however, by no means certain. In the number of the Journal referred to, Surgeon Perry states that of about a hundred autopsies during four years in Gibraltar, in cases of the so-called rock fever, in not one were the typical lesions of typhoid absent. On the other hand, it is held to be a fever due to chronic poisoning with fæcal emanations.

Fortunately, the mortality is not great. With reference to the treat-

* British Medical Journal, vol. i, 1889.

ment Bruce concludes that it should be directed principally to keeping the patient's strength up by fluid, easily digested food, by stimulants when required and by attention to ordinary hygienic principles. The removal of the patient from the infected area does not cut short the fever.

(5) MOUNTAIN FEVER.

Residence for a time at a high altitude is in some instances followed by a group of symptoms to which the term mountain sickness or mountain fever has been given. Several distinct diseases have undoubtedly been described. It is by no means certain that there is a special affection to which the term may be applied. An important group, the *mountain anæmia*, is associated with the *anchylostoma*, which has not yet been met with in this country. A second group of cases belongs unquestionably to typhoid fever, and undoubted instances of this disease occurring in mountainous regions in the West are referred to as mountain fever.

In the very full and clear report which Hoff * gives of five cases, the clinical picture is that of typhoid fever, and one of the patients died of perforation of the ileum with well-defined typhoid lesion. Even from the clinical reports, unless biased by notions of a rigidly characteristic picture of the disease, one might have said that all of Surgeon Hoff's cases were typhoid fever, and the post-mortem record leaves no question as to the nature of the malady. Woodward, commenting upon this communication, states that there is in the United States Army Medical Museum a second specimen from the case of so-called mountain fever contributed by Surgeon Girard.

Smart, who reviewed the entire question a few years ago, regarded the disease as a typho-malarial fever; but there is nothing in his account opposed to the opinion that it is a typhoid fever.

There is a third group to which, perhaps, alone the term mountain sickness should be applied—cases which present respiratory and cardiac symptoms, due to a high altitude. The pulse is rapid, there are giddiness, headache, sometimes nausea and vomiting, sensations of great prostration, and considerable respiratory distress. The original cases described by General Fremont were of this nature.

(6) MILIARY FEVER—SWEATING SICKNESS.

The disease is characterized by fever, profuse sweats, and an eruption of miliary vesicles. The disease prevailed and was very fatal in England in the fifteenth and sixteenth centuries, but of late years it has been confined entirely to certain districts in France (Picardy) and Italy. An epidemic of some extent occurred in France in 1887. Hirsch gives a

* American Journal of the Medical Sciences, January, 1880.

chronological account of 194 epidemics between 1718 and 1879, many of which were limited to a single village or to a few localities. Occasionally the disease has become widely spread. Slight epidemics have occurred in Germany and Switzerland. They are usually of short duration, lasting only for three or four weeks—sometimes not more than seven or eight days. As in influenza, a very large number of persons are attacked in rapid succession. In the mild cases there is only slight fever, with loss of appetite, an erythematous eruption, profuse perspiration, and an outbreak of miliary vesicles. The severe cases present the symptoms of intense infection—delirium, high fever, profound prostration, and hæmorrhage. The death-rate at the outset of the disease is usually high, and, as is so graphically described in the account of some of the epidemics of the middle ages, death may follow in a few hours.

SECTION II.

CONSTITUTIONAL DISEASES.

I. RHEUMATIC FEVER.

Definition.—An acute, non-contagious, febrile affection, depending probably upon an unknown infective agent, and characterized by multiple arthritis and a special tendency to involve the heart.

Etiology.—Acute rheumatism prevails in temperate and in humid climates. It is rare in the tropics. Statistics on the point are not available, but, judging from my own observations, I think that, in hospital practice at least, cases are much more frequent in England than in America. It prevails most extensively during the spring months. In Bell's statistics, of 456 cases treated at the Montreal General Hospital during ten years, the largest number of cases were admitted in February, March, and April. The same proportion seems true in Europe and in the cities of the Atlantic coast.

Age.—Young adults are most frequently affected, but the disease is by no means uncommon in children between the ages of ten and fifteen years. Sucklings are rarely affected, and probably many of the cases which have been described belong to a totally different affection, the arthritis of infants. In exceptional cases, however, true rheumatism does occur. The following age table is based upon 456 cases admitted to the Montreal General Hospital: Under 15 years, 4·38 per cent; from 15 to 25 years, 48·68 per cent; from 25 to 35 years, 25·87 per cent; from 35 to 45 years, 13·6 per cent; above 45 years, 7·4 per cent. Of the 655 cases analyzed by Whipham for the Collective Investigation Committee of the British Medical Association, only 32 cases occurred under the tenth year and 80 per cent between the twentieth and fortieth year. These figures scarcely give the ratio of cases in children.

Sex.—If all ages are taken, males are affected oftener than females. In the Collective Investigation Report there were 375 males and 279 females. Up to the age of twenty, however, females predominate. Between the ages of ten and fifteen girls are more prone to the disease.

Occupations which necessitate exposure to cold and to great changes

in temperature predispose strongly to rheumatism. We meet the disease oftenest in drivers, servants, bakers, sailors, and laborers. Heredity seems in some cases to have a special influence, and the disease is more common in certain families. Of all etiological factors, cold is believed to be the most potent. Many cases follow a sudden wetting or chilling of the skin.

The essential cause of rheumatism is still unknown. There are three chief theories:

(*a*) *Metabolic:* that it depends upon a morbid material produced within the system in defective processes of assimilation. It has been suggested that this material is lactic acid (Prout) or certain combinations with lactic acid (Latham). Our knowledge of the chemical relations of the various products produced in the regressive nutritive changes is too limited to base much reliance upon these views. Richardson claims to have produced rheumatism by injecting lactic acid and by its internal administration.

(*b*) The *nervous theory* advanced by J. K. Mitchell has many advocates. According to this view, either the nerve-centres are primarily affected by cold and the local lesions are really trophic in character, or the primary nervous disturbance leads to errors in metabolism and the accumulation of lactic acid in the system. The advocates of this view regard as analogous the arthropathies of myelitis, locomotor ataxia, and chorea.

(*c*) *Germ theory:* that the arthritis is due to a specific microbe. In favor of this view may be mentioned the close analogy which exists between rheumatism and certain of the infectious diseases. The analogy is marked with gonorrhœa, scarlet fever, and septic processes, which are frequently associated with arthritis and endocarditis. The investigations hitherto made have not, however, shown the constancy of any microorganism in the disease. Mantle and others have described micrococci in the blood, and several organisms have been found in the secondary inflammations of the disease, but none of them can be said to be specific or peculiar.

Morbid Anatomy.—There are no changes characteristic of the disease. The affected joints show hyperæmia and swelling of the synovial membranes and of the ligamentous tissues. There may be slight erosion of the cartilage. The fluid in the joint is turbid, albuminous in character, and contains leucocytes and a few fibrin flakes. Pus is very rare in uncomplicated cases. Rheumatism rarely proves fatal, except when there are serious complications, such as pericarditis, endocarditis, myocarditis, pleurisy, or pneumonia. The conditions found have nothing peculiar, nothing to distinguish them from other forms of inflammation. In death from hyperpyrexia no special changes occur. The blood usually contains an excessive amount of fibrin. In the secondary rheumatic inflammations, as pleurisy and pericarditis, various pus organisms have been found, possibly the result of a mixed infection.

Symptoms.—As a rule, the disease sets in abruptly, but it may be preceded by irregular pains in the joints, slight *malaise*, sore throat, and particularly by tonsillitis. A definite rigor is uncommon; more often there is slight chilliness. The fever rises quickly, and with it one or more of the joints become painful. Within twenty-four hours from the onset, the disease is fully developed. The temperature range is from 102° to 104°. The pulse is frequent, soft, and usually above 100. The tongue is moist, and rapidly becomes covered with a white fur. There are the ordinary symptoms associated with an acute fever, such as loss of appetite, thirst, constipation, and a scanty, highly acid, highly colored urine. In a majority of the cases there are profuse, very acid sweats, of a peculiar sour odor. Sudaminal and miliary vesicles are abundant. The mind is clear, except in the cases with hyperpyrexia. The affected joints are painful to move, and soon become swollen and hot, and present a reddish flush. The knees, ankles, elbows, and wrists are the joints usually attacked, not together, but successively. For example, if the knee is first affected, the redness may disappear from it as the wrists become painful and hot. The disease is seldom limited to a single articulation. The amount of swelling is variable. Extensive effusion into a joint is rare, and much of the enlargement is due to the infiltration of the periarticular tissues with serum. The swelling may be limited to the joint proper, but in the wrists and ankles it sometimes involves the sheaths of the tendons and produces great enlargement of the hands and feet. Corresponding joints are often affected. In attacks of great severity every one of the larger joints may be involved. The vertebral, sterno-clavicular, and phalangeal articulations are less often inflamed in acute than in gonorrhœal rheumatism. Perhaps no disease is more painful than acute polyarthritis. The inability to change the posture without agonizing pain, the drenching sweats, the prostration and utter helplessness, combine to make it one of the most distressing of febrile affections. A special feature of the disease is the tendency of the inflammation to subside in one joint while developing with great intensity in another.

The temperature range in an ordinary attack is between 102° and 104°. It is peculiarly irregular, with marked remissions and exacerbations, depending very much upon the intensity and extent of the articular inflammation. Defervescence is usually gradual. The profuse sweats materially influence the temperature curve. If a two-hourly chart is made and observations upon the sweats are noted, the remissions will usually be found coincident with the sweats. The perspiration is sour-smelling and acid at first; but, when persistent, becomes neutral or even alkaline.

The blood is profoundly and rapidly altered in acute rheumatism. There is, indeed, no acute febrile disease in which the anæmia develops with greater rapidity.

With the high fever a murmur may often be heard at the apex region. Endocarditis is also a common cause of an apex *bruit*. The heart

should be carefully examined at the first visit and subsequently each day.

The urine is, as a rule, reduced in amount, of high density and high color. It is very acid, and, on cooling, deposits urates. The chlorides may be greatly diminished or even absent. Febrile albuminuria is not uncommon.

The saliva may become acid in reaction and is said to contain an excess of sulphocyanides.

Subacute Rheumatism.

This represents a milder form of the disease, in which all the symptoms are less pronounced. The fever rarely rises above 101°; fewer joints are involved; and the arthritis is less intense. The cases may drag on for weeks or months, and the disease may finally become chronic. It should not be forgotten that in children this mild or subacute form may be associated with endocarditis or pericarditis.

Complications.—These are important and serious.

(1) **Hyperpyrexia.**—The temperature may rise rapidly a few days after the onset, and be associated with delirium; but not necessarily, for the temperature may rise to 108° or, as in one of Da Costa's cases, 110°, without cerebral symptoms. The delirium may precede or follow the onset of the hyperpyrexia. As a rule, with the high fever, the pulse is feeble and frequent, the prostration is extreme, and finally stupor supervenes.

(2) **Cardiac Affections.**—(*a*) *Endocarditis*, the most frequent and serious complication, occurs in a considerable percentage of all cases. The statistics upon this point are not of much value, as the diagnosis has been based, as a rule, upon the development of a systolic murmur at apex or base. This is quite untrustworthy; since it may depend upon causes other than endocarditis. The mitral segments are most frequently involved and the affection is usually of the simple, verrucose variety. Ulcerative endocarditis in the course of acute rheumatism is very rare. Of 209 cases of this disease which I analyzed, in only 24 did the symptoms of a severe endocarditis arise during the progress of acute or subacute rheumatism. This complication, in itself, is rarely dangerous. It produces few symptoms and is usually overlooked. Unhappily, though the valve at the time may not be seriously damaged, the inflammation starts changes which lead to sclerosis and retraction of the segments, and so to chronic valvular disease.

(*b*) *Pericarditis* may occur independently of or together with endocarditis. It may be simple fibrinous, sero-fibrinous, or in children purulent. Clinically we meet it more frequently in connection with rheumatism than all other affections combined. The physical signs are very characteristic. The condition will be fully described under its appropriate section. A peculiar form of delirium may develop during the progress of rheumatic pericarditis.

(*c*) *Myocarditis* is most frequent in connection with endo-pericardial changes. The anatomical condition is a granular or fatty degeneration of the heart-muscle, which leads to weakening of the walls and to dilatation. It is not, I think, nearly so common as the other cardiac affections. S. West has reported instances of acute dilatation of the heart in rheumatic fever, in one of which marked fatty changes were found in the heart-fibres.

(3) **Pulmonary Affections.**—Pneumonia and pleurisy are not uncommon, and frequently accompany the cases of endo-pericarditis. According to Howard's analysis of a large number of cases, there were pulmonary complications in only 10·5 per cent of cases of rheumatic endocarditis; in 58 per cent of cases of pericarditis; and in 71 per cent of cases of endo-pericarditis. Congestion of the lung is occasionally found, and in several cases has proved rapidly fatal.

(4) **Cerebral Complications.**—These are due, in part, to the hyperpyrexia and in part to the special action upon the brain of the toxic agent of the disease. They may be grouped as follows: (*a*) *Delirium.* This is usually associated with the hyperpyrexia, but may be independent of it. It may be active and noisy in character; more rarely a low muttering delirium, passing into stupor and coma. Special mention must be made of the delirium which occurs in connection with rheumatic pericarditis. Delirium, too, may be excited by the salicylate of soda, either shortly after its administration, or more commonly a week or ten days later. (*b*) *Coma*, which is more serious, may develop without preliminary delirium or convulsions, and may prove rapidly fatal. Certain of these cases are associated with hyperpyrexia; but Southey has reported the case of a girl who, without previous delirium or high fever, became comatose, and died in less than an hour. A certain number of such cases, as those reported by Da Costa, have been associated with marked renal changes and were evidently uræmic. The coma may develop during the attack, or after convalescence has set in. (*c*) *Convulsions* are less common, though they may precede the coma. Of 127 observations cited by Besnier, there were 37 of delirium, only 7 of convulsions, 17 of coma and convulsions, 54 of delirium, coma, and convulsions, and 3 of other varieties (Howard). (*d*) *Chorea.* The relations of this disease and rheumatism will be subsequently discussed. It is sufficient here to say that in only 88 out of 554 cases which I have analyzed from the Infirmary for Diseases of the Nervous System, Philadelphia, were chorea and rheumatism associated. It is most apt to develop in the slighter attacks in childhood. (*e*) *Meningitis* is extremely rare, though undoubtedly it does occur. It must not be forgotten that in ulcerative endocarditis, which is occasionally associated with acute rheumatism, meningitis is frequent.

(5) **Cutaneous Affections.**—Sweat-vesicles have already been mentioned as extremely common. A red miliary rash may also develop. Scarlatiniform eruptions are occasionally seen. Purpura, with or without urticaria,

may occur, and various forms of erythema. It is doubtful whether the cases of extensive purpura with urticaria and arthritis—peliosis rheumatica—belong truly to acute rheumatism.

(6) **Rheumatic Nodules.**—These curious structures, in the form of small subcutaneous nodules attached to the tendons and fasciæ, have been known for some years; but special attention has been paid to them of late, since their careful study by Barlow and Warner. They vary in size from a small shot to a large pea, and are most numerous on the fingers, hands, and wrists. They also occur about the elbows, knees, the spines of the vertebræ, and the scapulæ. They are not often tender. They do not necessarily come on during the fever, but may be found on its decline, or even independently altogether of an acute attack. They may develop with great rapidity and usually last for weeks or months. They are more common in children than in adults, and their presence may be regarded as a positive indication of rheumatism. They have been noted particularly in association with severe and chronic rheumatic endocarditis. They may occur in large numbers in adults, as in a case reported from my clinic in Philadelphia, by J. K. Mitchell. Histologically they are made up of round and spindle-shaped cells.

The *course* of acute rheumatism is extremely variable. It is, as Austin Flint first showed, a self-limited disease, and it is not probable that medicines have any special influence upon its *duration* or *course*. Gull and Sutton who likewise studied a series of sixty-two cases without special treatment arrived at the same conclusion.

Diagnosis.—Practically, the recognition of acute rheumatism is very easy; but there are several affections which, in some particulars, closely resemble it.

(1) **Multiple Secondary Arthritis.**—Under this term may be embraced the various forms of arthritis which come on or follow in the course of the infective diseases, such as gonorrhœa, scarlet fever, dysentery, and cerebro-spinal meningitis. Of these the gonorrhœal form will receive special consideration and is the type of the entire group.

(2) **Septic Arthritis,** which develops in the course of pyæmia from any cause, and particularly in puerperal fever. No hard and fast line can be drawn between these and the cases in the first group; but the inflammation rapidly passes on to suppuration and there is more or less destruction of the joints. The conditions under which the arthritis develops give a clew at once to the nature of the case. Under this section may also be mentioned:

(*a*) *Acute necrosis* or *acute osteo-myelitis*, occurring in the lower end of the femur, or in the tibia, and which may be mistaken for acute rheumatism. Sometimes, too, it is multiple. The greater intensity of the local symptoms, the involvement of the epiphyses rather than the joints, and the more serious constitutional disturbances are points to be considered. The condition is unfortunately often mistaken for acute arthritis, and, as

the treatment is essentially surgical, the error is one which may cost the life of the patient.

(*b*) *The acute arthritis of infants* must be distinguished from rheumatism. It is a disease which is usually confined to one joint (the hip or knee), the effusion in which rapidly becomes purulent. The affection is most common in sucklings and is undoubtedly pyæmic in character.* It may also develop in the gonorrhœal ophthalmia or vaginitis of the newborn, as pointed out by Clement Lucas.

(3) It is only in rare instances that gout and acute rheumatism are confounded. The localization in a single, usually a small, joint, the age, the history, the mode of onset—are features which enable us to recognize the cases readily.

Treatment.—The bed should have a smooth, soft, yet elastic mattress. The patient should wear a flannel night-gown, which may be opened all the way down the front and slit along the outer margin of the sleeves. Three or four of these should be made, so as to facilitate the frequent changes required after the sweats. He may wear also a light flannel cape about the shoulders. He should sleep in blankets, not in sheets, so as to reduce the liability to catch cold and obviate the unpleasant clamminess consequent upon heavy sweating. Chambers insisted that the liability to endocarditis and pericarditis was much reduced when the patients were in blankets.

Milk is the most suitable diet. It may be diluted with alkaline mineral waters. Lemonade and oatmeal or barley water should be freely given. The thirst is usually great and may be fully satisfied. There is no objection to broths and soups if the milk is not well borne. The food should be given at short and stated intervals. As convalescence is established a fuller diet may be allowed, but meat should be used sparingly.

The local treatment is of the greatest importance. It often suffices to wrap the affected joints in cotton. If the pain is severe, hot cloths may be applied, saturated with Fuller's lotion (carbonate of soda, 6 drachms; laudanum, 1 oz.; glycerine, 2 oz.; and water, 9 oz.). Tincture of aconite or chloral may be employed in an alkaline solution. Chloroform liniment is also a good application. Fixation of the joints is of great service in allaying the pain. I have seen, in a German hospital, the joints enclosed in plaster of Paris, apparently with great relief. Splints, padded and bandaged with moderate firmness, will often be found to relieve pain. Friction is rarely well borne in an acutely inflamed joint. Cold compresses are much used in Germany. The application of blisters above and below the joint, often relieves the pain. This method, which was used so much a few years ago, is not to be compared with the light application of the Paquelin thermo-cautery.

Medicines have little or no control over the duration or course of the

* Townsend, Acute Arthritis of Infants, American Journal of the Medical Sciences, January, 1890.

disease, which, like other self-limited affections, practically takes its own time to disappear. Salicyl compounds, which were regarded so long as specific in the disease, are now known to act chiefly by relieving pain. R. P. Howard's elaborate analysis shows that they do not influence the duration of the disease. Nor do they prevent the occurrence of cardiac complications, while under their use relapses are considerably more frequent than in any other method of treatment. In acute cases with severe pain the salicyl compounds give prompt relief and rarely disappoint us in their action. Sodium salicylate, in fifteen-grain doses for eight or ten doses, may be given. The bicarbonate of potassium in twenty-grain doses may be used with it. Many prefer salicin (gr. 20) in wafers; others the salicylic acid (gr. 20) or salol. I have for the past five or six years used the oil of wintergreen, recommended by Kinnicutt, and have found it quite as efficacious. Twenty minims may be given every two hours in milk. The salicyl compounds are best given in full doses at the outset of the disease, to relieve the pain. Then the dose should be reduced in frequency, or, if the symptoms have abated, stopped altogether, as relapses are certainly more frequent under their use.

Alkalies may be combined with the salicylates, or may be used alone. The potassium bicarbonate in half-drachm doses may be given every three or four hours until the urine is rendered alkaline. Fuller, who so warmly supported this method of treatment, was in the habit of ordering a drachm and a half of the sodium bicarbonate with half a drachm of potassium acetate in three ounces of water, rendered effervescent at the time of administration by half a drachm of citric acid or an ounce of lemon-juice. This is given every three or four hours, and usually by the end of twenty-four hours the urine is alkaline in reaction. The alkali is then reduced, and the amount subsequently regulated by the degree of acidity of the urine, only enough being given to keep the secretion alkaline. Opinion is almost unanimous that, under the alkaline treatment, cardiac complications are less common. The combination of the salicylates with the alkali is probably the most satisfactory. Care must be taken to watch the heart during the administration of these remedies. In the only fatal case of rheumatism which has come in my experience the patient had, owing to an error, taken the full first day's dose of Fuller's alkaline treatment for five successive days, instead of having the salt gradually reduced. She died suddenly on the fifth day after sitting up in bed. Salicylates also, if given largely, are very depressing to the circulation.

To allay the pain opium may be given in the form of Dover's powder, or morphia hypodermically. Antipyrin, antifebrin, and phenacetin are useful sometimes for the purpose. During convalescence iron is indicated in full doses, and quinine is a useful tonic. Of the complications, hyperpyrexia should be treated by the cold bath or the cold-pack. The treatment of endocarditis and pericarditis and the pulmonary complications will be considered under their respective sections.

II. CHRONIC RHEUMATISM.

Etiology.—This affection may follow an acute or subacute attack, but more commonly comes on insidiously in persons who have passed the middle period of life. In my experience it is extremely rare as a sequence of acute rheumatism. It is most common among the poor, particularly washer-women, day laborers, and those whose occupation exposes them to cold and damp.

Morbid Anatomy.—The synovial membranes are injected, but there is usually not much effusion. The capsule and ligaments of the joints are thickened, and the sheaths of the tendons in the neighborhood undergo similar alterations, so that the free play of the joint is greatly impaired. In long-standing cases the cartilages also undergo changes, and may show erosions. Even in cases with the severest symptoms, the joint may be very slightly altered in appearance. Important changes take place in the muscles and nerves adjacent to chronically inflamed joints, particularly in the mono-articular lesions of the shoulder or hip. Muscular atrophy supervenes partly from disuse, partly through nervous influences, either centric or reflex (Vulpian), or as a result of peripheral neuritis. In some cases when the joint is much distended the wasting may be due to pressure, either on the muscles themselves or on the vessels supplying them.

Symptoms.—Stiffness and pain are the chief features of chronic rheumatism. The latter is very liable to exacerbations, especially during changes in the weather. The joints may be tender to the touch and a little swollen, but seldom reddened. As a rule, many joints are affected; but there are instances in which the disease is confined to one shoulder, knee, or hip. The stiffness and pain are more marked after rest, and as the day advances the joints may, with exertion, become much more supple. The general health may not be seriously impaired. The disease is not immediately dangerous. Anchylosis may occur, and ultimately the joints may become very distorted. In many instances, particularly those in which the pain is severe, the general health may be seriously involved and the subjects become anæmic and very apt to suffer with neuralgia and dyspepsia. Valvular lesions, due to slow sclerotic changes, are not uncommon. They are associated with, not dependent upon, the articular disease.

The *prognosis* is not favorable, as a majority of the cases resist all methods of treatment. It is, however, a disease which persists indefinitely, and does not necessarily shorten life.

Treatment.—Internal remedies are of little service. It is important to maintain the digestive functions and to keep the general health at a high standard. Iodide of potassium, sarsaparilla, and guaiacum are sometimes beneficial. The salicylates are useless.

Local treatment is very beneficial. "Firing" with the Paquelin cautery relieves the pain, and it is perhaps the best form of counter-

irritation. Massage, with passive motion, helps to reduce swelling, and prevents anchylosis. It is particularly useful in cases which are associated with atrophy of the muscles. Electricity is not of much benefit. Climatic treatment is very advantageous. Many cases are greatly helped by prolonged residence in southern Europe or southern California. Rich patients should always winter in the South, and in this way avoid the cold, damp weather.

Hydrotherapeutic measures are specially beneficial in chronic rheumatism. Great relief is afforded by wrapping the affected joints in cold cloths, covered with a thin layer of blanket, and protected with oiled silk. The Turkish bath is useful, but the full benefit of this treatment is rarely seen except at bathing establishments. The hot alkaline waters are particularly useful, and a residence at the Hot Springs of Virginia or Arkansas, or at Banff, in the Rocky Mountains, on the Canadian Pacific Railway, will sometimes cure even obstinate cases.

III. PSEUDO-RHEUMATIC AFFECTIONS.

These are numerous, and occur as complications or sequelæ of many infectious diseases with which they have been considered. The one which is of most importance, and which, though a surgical affection, is usually treated of in works on medicine, is—

Gonorrhœal Rheumatism.—Though custom has sanctioned this term, the affection here considered has probably nothing whatever to do with rheumatism, but is an arthritis or synovitis of a septic nature, due to infection from the urethral discharge. It occurs either during an acute attack of gonorrhœa, or, more commonly, as the attack subsides, or when it has become chronic. It is far more frequent in men than in women. It is liable to recur, and is an affection of extraordinary obstinacy. It may involve many joints, but the knees and ankles are most commonly affected. It is peculiar in attacking certain joints which are rarely involved in acute rheumatism—as the sterno-clavicular, the intervertebral, the temporo-maxillary, and the sacro-iliac.

The *anatomical changes* are variable. The inflammation is often periarticular, and extends along the sheaths of the tendons. When effusion occurs in the joints it rarely becomes purulent. It has more commonly the characters of a synovitis. About the wrist and hand suppuration sometimes occurs in the sheaths. In the bacteriological examination the gonococci have been found in the exudate, but not invariably. They may be present in the tissues, however, and cause an effusion which may be sterile. It has been suggested that the simple arthritis or synovitis follows absorption of ptomaines from the urethral discharge, while the more severe suppurating forms are due to infection with pus organisms.

The *symptoms* of this disease are very variable. R. P. Howard recognized five clinical forms:

(*a*) *Arthralgic*, in which there are wandering pains about the joints, without redness or swelling. These persist for a long time.

(*b*) *Rheumatic*, in which several joints become affected, just as in subacute articular rheumatism. The fever is slight; the local inflammation may fix itself in one joint, but more commonly several become swollen and tender. In this form cerebral and cardiac complications may occur.

(*c*) *Acute gonorrhœal arthritis*, in which a single articulation becomes suddenly involved. The pain is severe, the swelling extensive, and due chiefly to peri-articular œdema. The general fever is not at all proportionate to the intensity of the local signs. The affection usually resolves, though suppuration occasionally supervenes.

(*d*) *Chronic Hydrarthrosis.*—This is usually mono-articular, and is particularly apt to involve the knee. It comes on often without pain, redness, or swelling. Formation of pus is rare. It occurred only twice in ninety-six cases tabulated by Nolen.

(*e*) *Bursal and Synovial Form.*—This attacks chiefly the tendons and their sheaths and the bursæ and the periosteum. The articulations may not be affected. The bursæ of the patella, the olecranon, and the tendo Achillis are most apt to be involved.

The disease is much more intractable than ordinary rheumatism, and relapses are extremely common. It may become chronic and last for years. A patient under my care, at the University Hospital, Philadelphia, was practically bedridden for nearly ten years with his first attack, and was carried from one health resort to another without getting much benefit. He finally recovered sufficiently to resume work, and enjoyed fair health for more than a year. Then he unfortunately had another attack of gonorrhœa. The multiple arthritis recurred, and when he came under my observation he had been ill nearly two years.

Complications.—Iritis is not infrequent and may recur with successive attacks. The visceral complications are rare. Endocarditis, pericarditis, and pleurisy may occur. R. L. MacDonnell recently analyzed twenty-seven cases of gonorrhœal rheumatism at the Montreal General Hospital, of which four presented signs of recent cardiac disease. Gluzinski has collected thirty-one cases from the literature. The endocarditis is usually simple, but occasionally there is an intense infection and ulcerative endocarditis with symptoms resembling typhoid fever.

Treatment.—The salicylates are of very little service, nor do they often relieve the pains in this affection. Iodide of potassium has also proved useless in my hands, even given in large doses. A general tonic treatment seems much more suitable—quinine, iron, and, in the chronic cases, arsenic.

The local treatment of the joints is very important. The thermocautery may be used to allay the pain and reduce the swelling. In acute

cases, fixation of the joints is very beneficial, and in the chronic forms, massage and passive motion. The surgical treatment of this affection, as carried out nowadays, is more satisfactory, and I have seen strikingly good results follow incision and irrigation.

IV. MUSCULAR RHEUMATISM (*Myalgia*).

Definition.—A painful affection of the voluntary muscles and of the fasciæ and periosteum to which they are attached. The affection has received various names, according to its seat, as torticollis, lumbago, pleurodynia, etc.

Etiology.—The attacks follow cold and exposure, the usual conditions favorable to the development of rheumatism. It is by no means certain that the muscular tissues are the seat of the disease. Many writers claim, perhaps correctly, that it is a neuralgia of the sensory nerves of the muscles. Until our knowledge is more accurate, however, it may be considered under the rheumatic affections.

It is most commonly met with in men, particularly those exposed to cold and whose occupations are laborious. It is apt to follow exposure to a draught of air, as from an open window in a railway carriage. A sudden chilling after heavy exertion may also bring on an attack of lumbago. Persons of a rheumatic or gouty habit are certainly more prone to this affection. One attack renders an individual more liable to another. It is usually acute, but may become subacute or even chronic.

Symptoms.—The affection is entirely local. The constitutional disturbance is slight, and, even in severe cases, there may be no fever. Pain is a prominent symptom. It may be constant, or may occur only when the muscles are drawn into certain positions. It may be a dull ache or a bruised pain, or sharp, severe, and cramp-like. It is often sufficiently intense to cause the patient to cry out. Pressure on the affected part usually gives relief. As a rule, myalgia is a transient affection, lasting from a few hours to a few days. Occasionally it is prolonged for several weeks. It is very apt to recur.

The following are the principal varieties:

(1) **Lumbago**, one of the most common and painful forms, affects the muscles of the loins and their tendinous attachments. It occurs chiefly in workingmen. It comes on suddenly, and in very severe cases completely incapacitates the patient, who may be unable to turn in bed or to rise from the sitting posture.

(2) **Stiff neck** or **torticollis** affects the muscles of the antero-lateral region of the neck. It is very common, and occurs most frequently in the young. The person holds the head in a peculiar manner, and rotates the whole body in attempting to turn it. Usually it is confined to one side. The muscles at the back of the neck may also be affected.

(3) **Pleurodynia** involves the intercostal muscles on one side, and in some instances the pectorals and serratus magnus. This is, perhaps, the most painful form of the disease, as the chest cannot be at rest. It is more common on the left than on the right side. A deep breath, or coughing, causes very intense pain, and the respiratory movements are restricted on the affected side. There may be pain on pressure, sometimes over a very limited area. It may be difficult to distinguish from intercostal neuralgia, in which affection, however, the pain is usually more circumscribed and paroxysmal, and there are tender points along the course of the nerves. It is sometimes mistaken for pleurisy, but careful physical examination readily distinguishes between the two affections.

(4) Among other forms which may be mentioned are **cephalodynia,** affecting the muscles of the head; **scapulodynia, omodynia,** and **dorsodynia,** affecting the muscles about the shoulder and upper part of the back. Myalgia may also occur in the abdominal muscles and in the muscles of the extremities.

Treatment.—Rest of the affected muscles is of the first importance. Strapping the side will sometimes completely relieve pleurodynia. No belief is more wide-spread among the public than the efficacy of porous plasters for muscular pains of all sorts, particularly those about the trunk. If the pain is severe and agonizing, a hypodermic of morphia gives immediate relief. For lumbago acupuncture is, in acute cases, the most efficient treatment. Needles of from three to four inches in length (ordinary bonnet-needles, sterilized, will do) are thrust into the lumbar muscles at the seat of the pain, and withdrawn after five or ten minutes. In many instances the relief is immediate, and I can corroborate fully the statements of Ringer, who taught me this practice, as to its extraordinary and prompt efficacy in many instances. The constant current is sometimes very beneficial. In many forms of myalgia the thermo-cautery gives great relief. In obstinate cases blisters may be tried. Hot fomentations are soothing, and at the outset a Turkish bath may cut short the attack. In chronic cases iodide of potassium may be used, and both guaiacum and sulphur have been strongly recommended. Persons subject to this affection should be warmly clothed, and avoid, if possible, exposure to cold and damp. In gouty persons the diet should be restricted and the alkaline mineral waters taken freely. Large doses of nux vomica are sometimes beneficial.

V. ARTHRITIS DEFORMANS (*Rheumatoid arthritis*).

Definition.—A chronic disease of the joints, characterized by changes in the cartilages and synovial membranes, with periarticular formation of bone and great deformity.

Etiology.—Long believed to be intimately associated both with gout

and rheumatism (whence the names rheumatic gout and rheumatoid arthritis), this close relationship seems now very doubtful, since in a majority of the cases no history of either affection can be determined. It is difficult to separate some cases from ordinary chronic rheumatism, but the multiple form has, in all probability, a nervous origin, as suggested by J. K. Mitchell. This view is based upon such facts as the association of the disease with shock, worry, and grief; the similarity of the arthritis to the arthropathies due to disease of the cord, as in locomotor ataxia; the symmetrical distribution of the lesions; the remarkable trophic changes which lead to alterations in the skin and nails, and occasionally to muscular wasting out of proportion to the joint mischief. Ord regards the disease as analogous to progressive muscular atrophy and due either to a primary lesion in the cord or to changes the result of peripheral irritation, traumatic, uterine, urethral, etc. The true nature of the disease is still obscure, but the neuro-trophic theory meets very many of the facts. Females are more liable to the disease than males. In Archibald E. Garrod's table of 500 cases there were 411 females and 89 males. It most commonly sets in between the ages of twenty and thirty, but it may begin as late as fifty. It occurs also in children; within the past five years there have been at my clinics four cases in children under twelve. The degree of deformity may be extreme even at this early age. Hereditary influences are not uncommon. In Garrod's cases there were in 216 instances a family history of joint disease. Seguin has reported the occurrence of three cases in children of the same family. It is stated that the disease is more common in families with phthisical history. It seems to be more frequent in women who have had ovarian and uterine trouble, or who are sterile. In this country acute rheumatism or gout in the forebears is rare. Mental worry, grief, and anxiety seem frequent antecedents. It is an affection quite as common in the rich as in the poorer classes, though in England and the continent the latter seem more prone to the disease. Though often attributed to cold or damp, and occasionally to injury, there is no evidence that these are efficient causes.

Morbid Anatomy.—The changes in the joints differ essentially from those of gout in the absence of deposits of urate of soda, and from chronic rheumatism by the existence of extensive structural alterations, particularly in the cartilages. We are largely indebted to the magnificent work of Adams for our knowledge of the anatomy of this disease. The changes begin in the cartilages and synovial membranes, the cells of which proliferate. The cartilage covering the joint undergoes a peculiar fibrillation, becomes soft, and is either absorbed or gradually thinned by attrition, thus laying bare the ends of the bone, which become smooth, polished, and eburnated. At the margins, where the pressure is less, the proliferating elements may develop into irregular nodules, which ossify and enlarge the heads of the bones, forming osteophytes which completely lock the joint. The periosteum may also form new bone. There is usu-

ally great thickening of the ligaments, and finally complete anchylosis results. This is rarely, however, a true anchylosis, but is caused by the osteophytes and thickened ligaments. There are often hyperostosis and increase in the articular ends of the bone in length and thickness. In long-standing cases and in old persons there may, on the other hand, be great atrophy of the heads of the affected bones. The spongy substance becomes friable, and in the hip-joint the wasting may reach such an extreme grade that the articulating surface lies between the trochanters. This is sometimes called *morbus coxæ senilis*. The anatomical changes may lead to great deformity. The metacarpal joints are enlarged and thickened, and the fingers are deflected toward the ulnar side. The toes often show a similar deflection.

The muscles become atrophied, and in some cases the wasting reaches a high grade. Neuritis has been demonstrated in the nerves about the joints.

Symptoms.—Charcot makes a convenient division of the cases into Heberden's nodosities, the general progressive form, and the partial or mono-articular form.

Heberden's Nodosities.—In this form the fingers are affected, and little hard nodules develop gradually at the sides of the distal phalanges. They are much more common in women than in men. They begin usually between the thirtieth and fortieth year. The subjects may be in perfect health, though more commonly they have digestive troubles, neuralgia, or rheumatic pains, or have had gout. Although these nodules are usually regarded as gouty, in many cases no manifestations of this disease occur. Heberden did not lay any stress upon the association. In the early stage the joints may be swollen, tender, and slightly red, particularly when knocked. The attacks of pain and swelling may come on in the joints at long intervals or follow indiscretion in diet. The little tubercles at the sides of the dorsal surface of the second phalanx increase in size, and give the characteristic appearance to the affection. The cartilages also become soft, and the ends of the bones eburnated. The condition is not curable; but there is this hopeful feature—the subjects of these nodosities rarely have involvement of the larger joints. They have been regarded, too, as an indication of longevity. Charcot states that in women with these nodes cancer seems more frequent.

General Progressive Form.—This occurs in two varieties, acute and chronic. The *acute* form may resemble, at its outset, ordinary articular rheumatism. There is involvement of many joints; swelling, particularly of the synovial sheaths and bursæ; not often redness; but there is moderate fever. Howard describes this condition as most frequent in young women from twenty to thirty years of age, often in connection with recent delivery, lactation, or rapid child-bearing. Acute cases may develop at the menopause. It may also come on in children. "These patients suffer in their general health, become weak, pale, depressed in spirits, and lose

flesh. In several cases of this form marked intervals of improvement have occurred; the local disease has ceased to progress, and tolerable comfort has been experienced perhaps until pregnancy, delivery, or lactation again determine a fresh outbreak of the disease."

The *chronic* form is by far the most common. The joints are usually involved symmetrically. The first symptoms are pain on movement and slight swelling, which may be in the joint itself or in the peri-articular sheaths. In some cases the effusion is marked, in others slight. The local conditions vary greatly, and periods of improvement alternate with attacks of swelling, redness, and pain. At first only one or two joints are affected; usually the joints of the hands, then the knees and feet; gradually other articulations are involved, and in extreme cases every articulation in the body is affected. Pain is an extremely variable symptom. Some cases proceed to the most extreme deformity without pain; in others the suffering is very great, particularly at night and during the exacerbations of the disease. There are cases in which pain of an agonizing character is an almost constant symptom, requiring for years the use of morphia.

Gradually the shape of the joints is greatly altered, partly by the presence of osteophytes, partly by the great thickening of the capsular ligaments, and still more by the retraction of the muscles. In moving the affected joint crepitation can be felt, due to the eburnation of the articular surfaces. Ultimately the joints become completely locked, not by a true bony anchylosis, but by the osteophytes which form around the articular surfaces, like ring-bone in horses. There is also a spurious anchylosis, caused by the thickening of the capsular ligaments and fibrous adhesions. The muscles about the joints undergo important changes. Atrophy from disuse gradually supervenes, and contractures tend to flex the thigh upon the abdomen and the leg upon the thigh. There are cases with rapid muscular wasting, symmetrical involvement of the joints, and trophic changes, which strongly suggest a central origin. Numbness, tingling, pigmentation or glossiness of the skin, and onychia may be present. In extreme cases the patient is completely helpless, and lies on one side with the legs drawn up, the arms fixed, and all the articulations of the extremities locked. Fortunately, it often happens in these severe general cases that the joints of the hand are not so much affected, and the patient may be able to knit or to write, though unable to walk or to use the arms. It is surprising indeed how much certain patients with advanced arthritis deformans can accomplish. No one who had seen the beautiful models and microscopic preparations of the late H. D. Schmidt, of New Orleans, could imagine that he had been afflicted for years with a most extreme grade of this terrible disease. In many cases, after involving two or three joints, the disease becomes arrested, and no further development occurs. It may be limited to the wrists, or to the knees and wrists, or to the knees and ankles. A majority of the patients finally reach a

quiescent stage, in which they are free from pain and enjoy excellent health, suffering only from the inconvenience and crippling necessarily associated with the disease.

Coincident affections are not uncommon. In the active stage the patients are often anæmic and suffer from dyspepsia, which may recur at intervals. There is no tendency to involvement of the heart.

The **partial** or **mono-articular** form affects chiefly old persons, and is seen particularly in the hip, the knee, the spinal column, or shoulder. It is, in its anatomical features, identical with the general disease. In the hip and shoulder the muscles early show wasting, and in the hip the condition ultimately becomes that already described as *morbus coxæ senilis*. These cases seem not infrequently to follow an injury. They differ from the polyarticular form in occurring chiefly in men and at a later period of life. One of the most interesting forms affects the vertebræ, completely locking the articulations, and producing the condition known as *spondylitis deformans*. When the cervical spine is involved the head cannot be moved up and down, but is carried stiffly. Usually rotation can be effected. The dorsal and lumbar spines may also be involved, and the body cannot be flexed in the slightest degree. No other joints may be affected.

Diagnosis.—Arthritis deformans can rarely be mistaken for either rheumatism or gout. It is important to distinguish from the mono-articular form the local arthritis of the shoulder-joint which is characterized by pain, thickening of the capsule and of the ligaments, wasting of the shoulder-girdle muscles, and sometimes by neuritis. This is an affection which is quite distinct from arthritis deformans, and is, moreover, in a majority of cases curable.

Treatment.—Arthritis deformans is an incurable disease. In many cases, after involvement of two or three joints, the progress is arrested. Too often it invades successively all the articulations, and in ten, fifteen, or twenty years the crippling becomes general and permanent.

The best that can be hoped for is a gradual arrest. It is useless to saturate the patients with iodide of potassium, salicylates, or quinine. Arsenic seems to do good as a general tonic. The improvement may be marked if large doses of it are given. Iron should be used freely, if there is anæmia. Careful attention to the digestion, plenty of good food, and fresh air are important measures. Hydrotherapy, with carefully performed massage, is best for the alleviation of the pain, and may possibly restrain the progress of the affection. In early cases local improvement and often great gain in the general strength follow a prolonged treatment at the hot mineral baths; but the practitioner should exercise care in recommending this mode of treatment, which is of very doubtful value when the disease is well established. I have repeatedly known cases to be rendered much worse by residence at these institutions. When good results, it is largely from change of scene and climate, and the careful

regulation of the diet. The local treatment is of benefit in arresting the progress. When there are much heat and pain the limb should be at rest, cold compresses applied at night, the joints wrapped in oiled silk, and in the morning thoroughly massaged. It is surprising how much can be done by carefully applied friction to reduce the thickening, to promote absorption of effusion, and to restore mobility. Massage is also of special benefit in maintaining the nutrition of the muscles, which early tend to atrophy. In the case of the knees this mode of treatment will sometimes prevent the retraction of the muscles and the gradual flexion of the legs on the thighs. No benefit can be expected from electricity.

VI. GOUT (*Podagra.*)

Definition.—A nutritional disorder, associated with an excessive formation of uric acid, and characterized clinically by attacks of acute arthritis, by the gradual deposition of urate of soda in and about the joints, and by the occurrence of irregular constitutional symptoms.

Etiology.—It is now generally recognized that the disease depends upon disturbed metabolism; most probably upon defective oxidation of nitrogenous food-stuffs.

Among important etiological factors in gout are the following:

(*a*) *Hereditary Influences.*—Statistics show that in from fifty to sixty per cent of all cases the disease existed in the parents or grandparents. The transmission is supposed to be more marked from the male side. Cases with a strong hereditary taint have been known to develop before puberty. The disease has been seen even in infants at the breast. Males are more subject to the disease than females. It rarely develops before the thirtieth year; and in a large majority of the cases the first manifestations appear before the age of fifty. (*b*) *Alcohol* is the most potent factor in the etiology of the disease. Fermented liquors favor its development much more than distilled spirits, and it prevails most extensively in countries like England and Germany, which consume the most beer and ale. Probably the greater tendency of malt liquors to induce gout is associated with the production of an acid dyspepsia. The lighter beers used in this country are much less liable to produce gout than the heavier English and Scotch ales. (*c*) *Food* plays a *rôle* equal in importance to that of alcohol. From the time of Hippocrates overeating has been regarded as a special predisposing cause. The excessive use of food, particularly of meats, disturbs gastric digestion and leads to the formation of lactic and volatile fatty acids. It is held by Garrod and others that these tend to decrease the alkalinity of the blood and to reduce its power of holding urates in solution. A special form of gouty dyspepsia has been described. A robust and active digestion is, however, often met in gouty persons. Gout is by no means confined to the rich. In England the combination of

poor food, defective hygiene, and an excessive consumption of malt liquors makes the "poor man's gout" a common affection. (*d*) *Lead.* Garrod has shown that workers in lead are specially prone to gout. In thirty per cent of his hospital cases the patients had been painters or workers in lead. The association is probably to be sought in the production by this poison of arterio-sclerosis and chronic nephritis. Something in addition is necessary, or certainly in this country we should more frequently see cases of the kind so common in London hospitals. Chronic lead-poisoning is here frequently associated with arterio-sclerosis and contracted kidneys, but acute arthritis is rare. Gouty deposits are, however, to be found in the big-toe joint and in the kidneys in these cases.

There are three theories with reference to gout:

(1) *The Uric-acid Theory.*—Sir Alfred Garrod, to whom the profession is indebted for so many careful studies in this disease, showed that there was an increase in the uric acid in the blood, due either to increased production or to diminished elimination; and that the alkalinity of the blood was also lessened. He attributes the deposition of the urate of soda to the diminished alkalinity of the plasma, which is unable to hold it in solution. An increase in the quantity of the uric acid produced, or any interference with elimination through the kidneys, may cause a sudden outbreak. The acute paroxysm is due to an accumulation of the urates in the blood, which he believes are responsible also for the preliminary dyspepsia, the coated tongue, the irritability of temper, and the general feelings of *malaise.* The sudden deposit of the crystalline urates about the joint leads to inflammation.

(2) *The Nervous Theory.*—The view of Cullen that gout was primarily an affection of the nervous system has been modified into a neuro-humoral view which has been advocated particularly by Sir Dyce Duckworth. On this theory there is a basic, arthritic stock—a diathetic habit, of which gout and rheumatism are two distinct branches. The gouty diathesis is expressed in (*a*) a neurosis of the nerve-centres, which may be inherited or acquired; and (*b*) "a peculiar incapacity for normal elaboration within the whole body, not merely in the liver or in one or two organs, of food, whereby uric acid is formed at times in excess, or is incapable of being duly transformed into more soluble and less noxious products" (Duckworth). The explosive neuroses and the influence of depressing circumstances, physical or mental, point strongly to the part played by the nervous system in the disease.

(3) *Ebstein's Theory.*—A nutritive tissue disturbance is the primary change leading to necrosis, and in the necrotic areas the urates are deposited. This is not unlike the view of Ord, who holds that there is a tendency, inherited or acquired, to a special form of tissue degeneration.

Morbid Anatomy.—The *blood* shows an excess of uric acid, as proved originally by Garrod. The uric acid may be obtained from the

blood-serum by the method known as uric-acid thread experiment, or from the serum obtained from a blister. To ʒ ij of serum add ♏v–vj of acetic acid in a watch-glass. A thread immersed in this will show in a few hours an incrustation of uric acid. This is not, however, peculiar to gout, but occurs in leukæmia and chlorosis. The important changes are in the articular tissues. The first joint of the great toe is most frequently involved; then the ankles, knees, and the small joints of the hands and wrists. The deposits may be in all the joints of the lower limbs and absent from those of the upper limbs (Norman Moore). If death takes place during an acute paroxysm, there are signs of inflammation, hyperæmia, swelling of the ligamentous tissues, and of effusion into the joint. The primary change, according to Ebstein, is a local necrosis, due to the presence of an excess of urates in the blood. This is seen in the cartilage and other articular tissues in which the nutritional currents are slow. In these areas of coagulation necrosis the reaction is always acid and the neutral urates are deposited in crystalline form, as insoluble acid urates. The articular cartilages are first involved. The gouty deposit may be uniform, or in small areas. Though it looks superficial, the deposit is invariably interstitial and covered by a thin lamina of cartilage. The deposit is thickest at the part most distant from the circulation. The ligaments and fibro-cartilage ultimately become involved and are infiltrated with chalky deposits, the so-called chalk-stones, or tophi. These are usually covered by skin; but in some cases, particularly in the metacarpophalangeal articulations, this ulcerates and the chalk-stones appear externally. The synovial fluid may also contain crystals. In very long-standing cases, owing to an excessive deposit, the joint becomes immobile. The marginal outgrowths in gouty arthritis are true exostoses (Wynne). The cartilage of the ear may contain tophi, which are seen as yellowish nodules at the margin of the helix. The cartilages of the nose, eyelids, and larynx are less frequently affected.

Of changes in the internal organs, those in the renal and vascular systems are the most important. The kidney changes believed to be characteristic of gout are: (*a*) A deposit of urates chiefly in the region of the papillæ. This is a less common change, however, than is usually supposed. Norman Moore found it in only twelve out of eighty cases. The apices of the pyramids show lines of whitish deposit. On microscopical examination the material is seen to be largely in the intertubular tissue. In some instances, however, the deposit seems to be both in the tissue and in the tubules. Ebstein, in his monograph, has described and figured areas of necrosis in both cortex and medulla, in the interior of which were crystalline deposits of urate of soda. The presence of these uratic concretions at the apices of the pyramids is not a positive indication of gout. They are not infrequent in this country, in which gout is rare. (*b*) An interstitial nephritis, either the ordinary "contracted kidney" or the arterio-sclerotic form, neither of which are in any way distinctive. It is

not possible to say in a given case that the condition has been due to gout unless marked evidences of the disease coexist.

The metatarso-phalangeal joint of the big toe should be carefully examined, as it may show typical lesions of gout without any outward token of arthritis.

Arterio-sclerosis is a very constant lesion. With it the heart, particularly the left ventricle, is found hypertrophied. According to some authors, concretions of urate of soda may occur on the valves.

Changes in the respiratory system are rare. Deposits have been found in the vocal cords, and uric-acid crystals have been met in the sputa of a gouty patient (J. W. Moore). Emphysema is a very constant condition in old cases.

Symptoms.—Gout is usually divided into acute, chronic, and irregular forms.

Acute Gout.—Premonitory symptoms are common—twinges of pain in the small joints of the hands or feet, nocturnal restlessness, irritability of temper, and dyspepsia. The urine is acid, scanty, and high-colored. It deposits urates on cooling, and there may be, according to Garrod, transient albuminuria. There may be traces of sugar (gouty glycosuria). Before an attack the output of uric acid is low and is also diminished in the early part of the paroxysm. In some instances the throat is sore, and there may be asthmatic attacks. The attack sets in usually in the early morning hours. The patient is aroused by a severe pain in the metatarso-phalangeal articulation of the big toe, and more commonly on the right than on the left side. The pain is agonizing, the joint swells rapidly, and becomes hot, tense, and shiny. The sensitiveness is extreme, and the patient describes the pain as if the joint were being pressed in a vice. There is fever, and the temperature may rise to 102° or 103°. Toward morning the severity of the symptoms subsides, and, although the joint remains swollen, the day may be passed in comparative comfort. The symptoms recur the next night, and the fit, as it is called, usually lasts for from five to eight days, the severity of the symptoms gradually abating. Occasionally other joints are involved, particularly the big toe of the opposite foot. The inflammation, however intense, never goes on to suppuration. With the subsidence of the swelling the skin desquamates. After the attack the general health may be much improved. Recurrences are frequent. Some patients have three or four attacks in a year; others at longer intervals. Lecorché has shown that the amount of uric acid is reduced prior to an attack, diminishes during the first two days, then increases very much and falls toward the close.

The term *retrocedent* or *suppressed* gout is applied to serious internal symptoms, coincident with a rapid disappearance or improvement of the local signs. Very remarkable manifestations may occur under these circumstances. The patient may have severe gastro-intestinal symptoms—pain, vomiting, diarrhœa, and great depression—and death may occur

during such an attack. Or there may be cardiac manifestations—dyspnœa, pain, and irregular action of the heart. In some instances in which the gout is said to attack the heart, an acute pericarditis develops and proves fatal. So, too, there may be marked cerebral manifestations—delirium and coma, and even apoplexy—but in a majority of these instances the symptoms are, in all probability, uræmic.

Acute gout is a rare disease in America, and in hospital practice is almost unknown. Among the well-to-do and even among club-men—a class particularly liable—it is infrequent, in comparison with the prevalence in the corresponding classes in England. Men in large family practice may pass a year or more without seeing a case. It has become more common, however, during the past twenty-five years.

Chronic Gout.—With increased frequency in the attacks, the articular symptoms persist for a longer time, and gradually many joints become affected. Deposits of urates take place, at first in the articular cartilages and then in the ligaments and capsular tissues; so that in the course of years the joints become swollen, irregular, and deformed. The feet are usually first affected, then the hands. In severe cases there may be extensive concretions about the elbows and knees and along the tendons and in the bursæ. The tophi appear in the ears. Finally, a unique clinical picture is produced which cannot be mistaken for any other affection. The skin over the tophi may rupture or ulcerate, and about the knuckles the chalk-stones may be freely exposed. Patients with chronic gout are usually dyspeptic, often of a sallow complexion, and show signs of arteriosclerosis. The pulse tension is increased, the vessels are stiff, and the left ventricle is hypertrophied. The urine is increased in amount, is of low specific gravity, and usually contains a slight amount of albumen, with a few hyaline casts. Patients with chronic gout may show remarkable mental and even bodily vigor. Certain of the most distinguished members of our profession have been terrible sufferers from this disease—notably the elder Scaliger, Jerome Cardan, and Sydenham, whose statement that "more wise men than fools are victims of the affection" still holds good.

Irregular Gout.—This is a motley, ill-defined group of symptoms, manifestations of a condition of disordered nutrition, to which the terms *gouty diathesis* or *lithæmic state* have been given. Cases are seen in members of gouty families, who may never themselves have suffered from the acute disease, and in persons who have lived not wisely but too well, who have eaten and drunk largely, lived sedentary lives, and yet have been fortunate enough to escape an acute attack. It is interesting to note the various manifestations of the disease in a family with marked hereditary disposition. The daughters often escape, while one son may have gouty attacks of great severity, even though he lives a temperate life and tries in every way to avoid the conditions favoring the disorder. Another son has, perhaps, only the irregular manifestations and never the acute articu-

lar affection. While the irregular features are perhaps more often met with in the hereditary affection, they are by no means infrequent in persons who appear to have acquired the disease. The tendency in some families is to call every affection gouty. Even infantile complaints, such as scald-head, naso-pharyngeal vegetations, and enuresis, are often regarded, without sufficient grounds, I believe, as evidences of the family ailment. Among the commonest manifestations of irregular gout are the following:

(*a*) *Cutaneous Eruptions.*—Garrod and others have called special attention to the frequent association of eczema with the gouty habit. The French in particular insist upon the special liability of gouty persons to skin affections, the *arthritides*, as they call them.

(*b*) *Gastro-intestinal Disorders.*—Attacks of what is termed biliousness, in which the tongue is furred, the breath foul, the bowels constipated, and the action of the liver torpid, are not uncommon in gouty persons.

(*c*) *Cardio-vascular Symptoms.*—With the lithæmia, arterio-sclerosis is frequently associated. The blood tension is persistently high, the vessel walls become stiff, and cardiac and renal changes gradually develop. In this condition the manifestations may be renal, as when the albuminuria becomes more marked, or dropsical symptoms supervene. The manifestations may be cardiac, when the hypertrophy of the left ventricle fails and there are palpitation, irregular action, and ultimately a condition of asystole. Or, finally, the manifestations may be vascular, and involvement of the coronary arteries may cause sudden death. Aneurism may develop and prove fatal, or, as most frequently happens, a blood-vessel gives way in the brain, and the patient dies of apoplexy. It makes but little difference whether we regard this condition as primarily an arterio-sclerosis or as a gouty nephritis; the point to be remembered is that the nutritional disorder with which an excess of uric acid is associated induces in time increased tension, arterio-sclerosis, chronic interstitial nephritis, and changes in the myocardium. Pericarditis is not infrequent in connection with the granular kidney met with in gout.

(*d*) *Cerebral Manifestations.*—Headache is frequent. Haig has called special attention to the association of this symptom with retention of uric acid in the system. Neuralgias are not uncommon; sciatica and paræsthesias may develop. A common gouty manifestation, upon which Duckworth has laid stress, is the occurrence of hot or itching feet at night. Cramps in the legs may also be very troublesome. Hutchinson has called attention to hot and itching eyeballs as a frequent sign of masked gout. More serious cerebral manifestations result from a condition of arterio-sclerosis. Apoplexy is a common termination of gout. A low meningitis may develop, usually basilar.

(*e*) *Urinary Disorders.*—The urine is highly acid and high-colored, and may deposit on standing crystals of lithic acid. Transient and tem-

porary increase in this ingredient cannot be regarded as serious. In many cases of chronic gout the amount may be diminished, and only increased at certain periods, forming the so-called uric-acid showers. Sugar is found intermittently in the urine of gouty persons—gouty glycosuria. It may pass into true diabetes, but is usually very amenable to treatment. Oxaluria may also be present Gouty persons are specially prone to calculi, Jerome Cardan to the contrary, who reckoned freedom from stone among the chief of the *dona podagræ*. Minute quantities of albumen are very common in persons of gouty dyscrasia, and, when the renal changes are well established, tube-casts. Urethritis, accompanied with a well-marked purulent discharge, may develop, so it is stated, usually at the end of an attack. It may occur spontaneously, or follow a pure connection.

(*f*) *Pulmonary Disorders.*—There are no characteristic changes, but, as Greenhow has pointed out, chronic bronchitis occurs with great frequency in persons of a gouty habit.

(*g*) Of eye affections, iritis, glaucoma, hæmorrhagic retinitis, and suppurative panopthalmitis have been described.

Treatment.—Individuals who have inherited a tendency to gout, or who have shown any manifestations of it, should live temperately, abstain from alcohol, and eat moderately. An open-air life, with plenty of exercise and regular hours, does much to counteract an inborn tendency to the disease.

Diet.—Experience has shown that a modified nitrogenous diet is the most suitable. Starchy and saccharine articles of food are to be taken in very limited quantities; as "the conversion of azotized food is more complete with a minimum of carbohydrates than it is with an excess of them —in other words, one of the best means of avoiding the accumulation of lithic acid in the blood is to diminish the carbohydrates rather than the azotized foods" (Draper). Meats of all kinds, except perhaps the coarser sorts, such as pork and veal, and salted provisions, may be used. Eggs, oysters, and fish may be taken. Lobsters and crabs, particularly when made into salads, are to be eschewed. The sugar should be reduced to a minimum. The sweeter fruits should not be taken. Oranges and lemons may be allowed. Strawberries, bananas, and melons should not be eaten. If necessary, saccharin may be substituted for cane sugar. Potatoes should be sparingly used. The fresh vegetables, such as lettuce, cucumbers, tomatoes, and cauliflower, may be taken freely. Hot rolls and cakes of all sorts, hominy, grits, and the more starchy forms of prepared foods are not suitable. The various articles of diet prepared from corn should be avoided. Fats are easily digested and may be taken freely. In obstinate cases great benefit is derived from an exclusively milk diet.

Persons with a gouty tendency should be encouraged to drink freely of such mineral waters as they prefer. They keep the interstitial circulation active and so favor elimination. Milk and potash-water form a pleasant and wholesome drink for a lithæmic patient. Alcohol in all forms

should be avoided. When from any cause a stimulant is indicated, claret, dry sherry, or good whisky is preferable. Champagne is particularly pernicious. Persons with a marked tendency to lithæmia should be urged to restrict the appetite and to take only a moderate amount of food. Overeating is not far behind excessive drinking in its injurious effects. Indeed, a majority of people over forty years of age take more food than is required to maintain the equilibrium of health. Gout, in many cases, is evidence of an overfed, overworked, and consequently clogged machine.

The skin should be kept active: if the patient is robust, by the morning cold bath with friction after it; but if weak or debilitated, the evening warm bath should be substituted. An occasional Turkish bath with active shampooing is advantageous. The secretion of urine should be fully maintained, and the specific gravity reduced by diluents to at least 1·015. The bowels should be kept open and an occasional saline purgative may be administered. The patient should dress warmly, avoid rapid alterations in temperature, and be careful not to have the skin suddenly chilled. Gouty persons derive much benefit from taking certain waters, such as Saratoga, the Bedford, the White Sulphur of Virginia, in this country; the Bath and Harrogate, in England; and those of Carlsbad, Kissingen, Homburg, Vichy, and Contrexéville, on the continent.

In an acute attack the limb should be elevated and the affected joint wrapped in cotton-wool. Warm fomentations, or Fuller's lotion, may be used. Steaming the joint is sometimes beneficial. A brisk mercurial purge is always advantageous at the outset. The wine or tincture of colchicum, in doses of twenty to thirty minims, may be given every four hours in combination with the citrate of potash or the citrate of lithium. The colchicum should be carefully watched. It has, in a majority of the cases, a powerful influence over the symptoms; relieving the pain and reducing, sometimes with great rapidity, the swelling and redness. It should be promptly stopped as soon as it has relieved the pain. In cases in which the pain and sleeplessness are more distressing and do not yield to colchicum, morphia may be necessary. The patient should be placed on a low diet, chiefly of milk and barley-water, but if there is any debility, strong broths may be given, or eggs. It is occasionally necessary to give small quantities of stimulants. Potash water, Apollinaris, or Seltzer water should be taken freely. Waters with the sodium salts should be avoided. During convalescence meats and fish and game may be taken, and gradually the patient may resume the diet previously laid down.

In the chronic and irregular forms of gout the treatment by hygiene and diet is most suitable. Colchicum is not often required, though in small doses it is sometimes beneficial. Lithium salts do good, since a combination of uric acid with lithium is more soluble than the sodium salt. There is no good native lithia water. The medicine is best given

in potash water, in a glassful of which five grains of the citrate of lithium may be taken three times a day, or the *liquor lithiæ effervescens* of the British Pharmacopœia may be used. The mineral waters above mentioned are particularly beneficial, partly in themselves, and partly owing to the strict regulations to which the patient is subjected when taking the "cure." Ammoniacum, guaiacum, and preparations of quinine and iron are sometimes serviceable in the chronic gout. Iodide of potassium and the benzoates are also recommended. The local treatment of joints affected with chronic gout is not satisfactory. Hydrotherapeutic measures, the Paquelin, and massage may be tried.

VII. DIABETES MELLITUS.

Definition.—A disorder of nutrition, in which sugar accumulates in the blood and is excreted in the urine, the daily amount of which is greatly increased.

Etiology.—Hereditary influences play an important *rôle*, and cases are on record of its occurrence in many members of the same family. There are instances of the coexistence of the disease in husband and wife. Men are more frequently affected than women. It is a disease of adult life; a majority of the cases occur from the third to the sixth decade. It is rare in childhood, but cases are on record in children under one year of age. Persons of a neurotic temperament are often affected. It is a disease of the higher classes. Hebrews seem especially prone to it; one fourth of Frerichs' cases were of the Semitic race. In a considerable proportion of the cases of diabetes the subjects have been excessively fat at the beginning of, or prior to, the onset of the disease. It must be remembered, however, that a slight trace of sugar is not very uncommon in obese persons. This so-called lipogenic glycosuria is not of grave significance, and is only occasionally followed by true diabetes. There are instances on record in which obesity with diabetes has occurred in three generations. It is more common in cities than in country districts. Gout, syphilis, and malaria have been regarded as predisposing causes. Mental shock, severe nervous strain, and worry precede many cases. The combination of intense application to business, over-indulgence in food and drink, with a sedentary life, seem particularly prone to induce the disease. It may set in during pregnancy, and in rare instances may only occur at this period. Injury to or disease of the spinal cord or brain has been followed by diabetes. In the carefully analyzed cases of Frerichs there were thirty instances of organic disease of these parts. The medulla is not always involved. In only four of his cases, which showed organic disease, was there sclerosis or other anomaly of this part. An irritative lesion of Bernard's diabetic centre in the medulla is an occasional cause. I saw with Riess, at the Friedrichshain, Berlin, a woman who had anomalous

cerebral symptoms and diabetes, and in whom there was found post mortem a cysticercus in the fourth ventricle.

Of late years lesions of the pancreas have been held to cause diabetes, and in a certain number of cases this organ is affected. The disease has occasionally followed the infectious fevers. A few cases have followed injury without involvement of the brain or cord.

In comparison with European countries diabetes is a rare disease in America. The last census gave only 2·8 per one hundred thousand of population, against a ratio of from five to nine in the former. In this region the incidence of the disease may be gathered from the fact that among thirty-five thousand patients under treatment at the Johns Hopkins Hospital and Dispensary there were only ten cases.

We are ignorant of the nature of the disease. Normally the carbohydrates taken with the food are stored in the liver as glycogen, and then utilized as needed by the system. Glycogen can also be formed from the proteids of the food, and under certain circumstances sugar may be directly formed from the body proteids. Whenever the sugar in the systemic blood exceeds a definite amount it is discharged by the kidneys, producing glycosuria. Theoretically the condition may be supposed to be induced by:

(*a*) The ingestion of a larger quantity of carbohydrates and peptones than can be warehoused, so to speak, in the liver as glycogen, so that part has to pass over into the hepatic blood. Some of the instances of lipogenic or dietetic glycosuria are of this nature.

(*b*) Disturbances of the liver function: (1) Changes in the circulation under nervous influences. Puncture of the medulla, lesions of the cord, and central irritation of various kinds are followed by glycosuria, which is attributed to a vaso-motor paralysis (more rapid blood-flow) induced by these causes. On this view the disease is a neurosis. (2) Instability of the glycogen, owing either to imperfect formation or to conditions of the cells which render it less stable. Phloridzin and other substances which cause diabetes very probably act in this way.

(*c*) Defective assimilation of the glucose in the system. How and under what normal circumstances the sugar is utilized we do not yet know. Theoretically faulty metabolism would explain the condition.

Interesting observations have of late made it probable that the pancreas may in some cases be the seat of the trouble. Lesions of this organ have frequently been met with in diabetes. Von Mering and Minkowski have shown that extirpation of the gland in dogs is followed by glycosuria, but, if a small portion remains, sugar does not appear in the urine, facts which have been confirmed by Lepine and others. The pancreas, on this view, has, like the liver, a double secretion—an external, which is poured into the intestines, and an internal, which passes into the blood. This latter is supposed to be of the nature of a ferment, in the presence of which alone the normal assimilative processes can take place with the glycogen. Disease of the pancreas causes diabetes by preventing the formation of

the glycolytic ferment. Even when, as in a majority of instances of diabetes, the organ is apparently normal, a functional trouble may disturb the formation of this ferment. The fact that if a small portion of the gland is left, in the experiments upon dogs, diabetes does not occur, is analogous to the remarkable circumstance that a small fragment of the thyroid is sufficient to prevent the development of artificial myxœdema. It has recently been stated by Falkenberg that extirpation of the thyroid gland in dogs is also followed by diabetes.

Morbid Anatomy.—Saundby * has recently analyzed the changes which occur in this disease.

The *nervous system* shows no constant lesions. In a few instances there have been tumors or sclerosis in the medulla, or, as in the case above mentioned, a cysticercus has pressed on the floor. Cysts have been met with in the white matter of the cerebrum and perivascular changes have been described. Glycogen has been found in the spinal cord. In the peripheral nervous system there are instances in which tumors have been found pressing on the vagus. A secondary multiple neuritis is not rare, and to it the so-called diabetic tabes is probably due.

In the sympathetic system the ganglia have been enlarged and in some instances sclerosed, but there is nothing peculiar in these changes. The *blood* may contain as high as 0·4 per cent of sugar instead of 0·15 per cent. The plasma is usually loaded with fat, the molecules of which may be seen as fine particles. When drawn, a white creamy layer coats the coagulum, and there may be lipæmic clots in the small vessels. There are no special changes in the red or white corpuscles. Gabritschewsky has shown that the "polynuclear" leucocytes in diabetes contain glycogen. Glycogen can occur in normal blood, but it is here extra-cellular. It has been also found in the polynuclear leucocytes in leukæmia. The *heart* shows no characteristic changes. Endocarditis is very rare. The *lungs* show important changes. Acute broncho-pneumonia or croupous pneumonia (either of which may terminate in gangrene) and tuberculosis are common. The so-called diabetic phthisis is always tuberculous and results from a caseating broncho-pneumonia. In rare cases there is a chronic interstitial pneumonia, non-tuberculous. Fatty embolism of the pulmonary vessels has been described in connection with diabetic coma.

The *liver* is usually enlarged, fatty degeneration is common, and French writers have described a form of cirrhosis. Letulle, who has described remarkable examples of this so-called diabetic cirrhosis—the *cirrhose pigmentaire*—thinks the change is due to abnormal destruction of the blood-cells. It may be associated with bronzing of the skin.

The *pancreas*, as pointed out by Lancereaux, shows important changes. Saundby states that in seven out of fifteen cases it was atrophied, abnor-

* Bradshaw Lecture, Royal College of Physicians of London, 1890; and Lectures on Diabetes, E. B. Treat, New York, 1891.

mally firm and fibroid in four, and normal in only four. A patient of W. T. Bull died of diabetes after extirpation of the pancreas. In some instances there is a pigmentary cirrhosis analogous to that which occurs in the liver, and this induration seems to be an important change. Cancer of the pancreas has been met with, and Longstreth found, in one instance, cystic disease. Fat necrosis of the pancreas has also been found. Neither the *stomach* nor the *intestines* show any characteristic lesions.

The *kidneys* are sometimes fatty, and show a hyaline change in the tubular epithelium, particularly in the descending limb of the loop of Henle. It also occurs in the capillary vessels. Saundby confirms the occurrence of this hyaline change, and its restriction to the epithelium of Henle's tubes.

Symptoms.—*Acute* and *chronic* forms are recognized, but there is no essential difference between them, except that in the former the patients are younger, the course more rapid, and the emaciation more marked.

It is also possible to divide the cases into (1) *lipogenic* or *dietetic*, which includes the transient glycosuria of stout persons; (2) *neurotic*, due to injuries or functional disorders of the nervous system; and (3) *pancreatic*, in which there is a lesion of the pancreas. It is, however, by no means easy to discriminate in all cases between these forms. Of late attempts have been made to separate a clinical variety analogous to experimental pancreatic diabetes. Hirschfeld, from Guttmann's clinic, has described cases running a rapid and severe course usually in young and middle-aged persons. The polyuria is less common or even absent, and there is a striking defect in the assimilation of the albuminoids and fats, as shown by the examination of the fæces and urine. In four of seven cases autopsies were made and the pancreas was found atrophic in two, cancerous in one, and in the fourth exceedingly soft.

The *onset* of the disease is gradual and either frequent micturition or inordinate thirst first attracts attention. Very rarely it sets in rapidly, after a sudden emotion, an injury, or after a severe chill. When fully established the disease is characterized by great thirst, the passage of large quantities of saccharine urine, a voracious appetite, and, as a rule, progressive emaciation.

The Urine.—The amount varies from six or eight pints in mild cases to thirty or forty pints in very severe cases. In rare instances the quantity of urine is not much increased. Under strict diet the amount is much lessened, and in intercurrent febrile affections it may be reduced to normal. The specific gravity is high, ranging from 1·025 to 1·045. The urine is pale in color, almost like water, and has a sweetish odor and a distinctly sweetish taste. The reaction is acid. Sugar is present in varying amounts. In mild cases it does not exceed one and a half or two per cent, but it may reach from five to ten per cent. The total amount excreted in the twenty-four hours may range from ten to twenty ounces, and in

exceptional cases from one to two pounds. The following are the most satisfactory tests:

Fehling's Test.—The solution consists of sulphate of copper (grs. 90½), neutral tartrate of potash (grs. 364), solution of caustic soda (fl. ozs. 4), and distilled water to make up six ounces. Put a drachm of this in a test-tube and boil (to test the reagent); add an equal quantity of urine and boil again, when, if sugar is present, the yellow suboxide of copper is thrown down. The solution must be freshly prepared, as it is apt to decompose.

Trommer's Test.—To a drachm of urine in a test-tube add a few drops of a dilute sulphate-of-copper solution and then as much *liquor potassæ* as urine. On boiling, the copper is reduced if sugar be present, forming the yellow or orange-red suboxide. There are certain fallacies in the copper tests. Thus, a substance called glycuronic acid is met with in the urine after the use of certain drugs—chloral, phenacetin, morphia, chloroform, etc.—which reduces copper. It has been found in the urine of an apparently healthy man (Ashdown).

Fermentation Test.—This is free from all doubt. Place a small fragment of yeast in a test-tube full of urine, which is then inverted over a glass vessel containing the same fluid. If sugar is present, fermentation goes on with the formation of carbon dioxide, which accumulates in the upper part of the tube and gradually expels the urine.*

Of other ingredients in the urine, the urea is increased, the uric acid does not show special changes, and the phosphates may be greatly in excess. Ralfe has described a great increase in the phosphates, and in some of these cases, with an excessive excretion, the symptoms may be very similar to those of diabetes, though the sugar may not be constantly present. The term phosphatic diabetes has sometimes been applied to them. *Acetone* and acetone-forming substances are not infrequently present. Le Nobel's test for acetone is as follows: "Pour an ounce of urine into a urine glass; add a drachm or two of nitro-prusside of sodium (five grains to one ounce) and a few drops of strong liquid ammonia. After standing a few minutes a rose-violet color is developed, which, if much acetone is present, may require diluting with water in order to bring out the brilliancy of its color" (Saundby).

Glycogen has also been described as present in the urine.

Albumen is not infrequent. It occurred in nearly thirty-seven per cent of the examinations made by Lippman at Carlsbad.

Among the *general symptoms* of the disease, *thirst* is one of the most distressing. A very large amount of water is required to keep the sugar in solution and for its excretion in the urine. The amount of water consumed will be found to bear a definite ratio to the quantity excreted. Instances, however, are not uncommon of pronounced diabetes in which the

* For quantitative and other tests the student is referred to Tyson, On the Urine, or the standard works on urinalysis.

thirst is not excessive; but in such cases the amount of urine passed is never large. The thirst is most intense an hour or two after meals. As a rule, the digestion is good and the appetite inordinate. A story is told of a man with diabetes who was paid to stay away from a certain restaurant at which dinners were given at fixed prices. It is sometimes impossible to satiate the ravenous appetite of a diabetic patient. The condition is sometimes termed *bulimia* or *polyphagia*.

The tongue is usually dry, red, and glazed, and the saliva scanty. The gums may become swollen, and in the later stages aphthous stomatitis is common. Constipation is the rule.

In spite of the enormous amount of food consumed a patient may become rapidly emaciated. This loss of flesh bears some ratio to the polyuria, and when, under suitable diet, the sugar is reduced, the patient may quickly gain in flesh. The skin is dry and harsh, and perspirations rarely occur, except when phthisis coexists. Drenching sweats have been known to alternate with excessive polyuria. The temperature is often subnormal; the pulse is usually frequent, and the tension increased. Many diabetics, however, do not show marked emaciation. Patients past the middle period of life may have the disease for years without much disturbance of the health, and may remain well nourished. These are the cases of the *diabète gras* in contradistinction to *diabète maigre*.

Diabetes in Children.—Recently Stern has analyzed 117 cases in children. They usually occur among the better classes. Six were under one year of age. Hereditary influences were marked. The course of the disease is, as a rule, much more rapid than in adults. The shortest duration was two days. In seven cases it did not last a month. One case is mentioned of a child apparently born with the glycosuria, who recovered in eight months.

Complications.—(*a*) **Cutaneous.**—Boils and carbuncles are extremely common. Eczema is also met with and at times an intolerable itching. In women the irritation of the urine may cause the most intense pruritus pudendi, and in men a balanitis. Rarer affections are xanthoma and purpura. Gangrene is not uncommon. William Hunt has analyzed 64 cases. In 50 the localities were as follows: Feet and legs, 37; thigh and buttock, 2; nucha, 2; external genitals, 1; lungs, 3; fingers, 3; back, 1; eyes, 1. Perforating ulcer of the foot may occur.

(*b*) **Pulmonary.**—The patients are not infrequently carried off by *acute pneumonia*, which may be lobar or lobular. *Gangrene* is very apt to supervene, but the breath does not necessarily have the foul odor of ordinary gangrene.

Tuberculous broncho-pneumonia is very common. It was formerly thought, from its rapid course and the limitation of the disease to the lung, that this was not a true tuberculous affection; but in the cases which have come under my notice bacilli have been present, and the condition is now generally regarded as tuberculous.

(*c*) **Renal.**—*Albuminuria* is a tolerably frequent complication. The amount varies greatly, and, when slight, does not seem to be of much moment. It is sometimes associated with arterio-sclerosis. It occasionally precedes the development of the diabetic coma. Occasionally cystitis develops.

(*d*) **Nervous System.**—(1) *Diabetic coma*, first studied by Küssmaul, is the most serious complication of the disease, and carries off a considerable proportion of all cases, particularly in the young. It may occur when diabetes is unsuspected, as in two cases recently reported by Francis Minot. Frerichs recognized three groups of cases: (α) Those in which after exertion the patients were suddenly attacked with weakness, syncope, somnolence, and gradually deepening unconsciousness; death occurring in a few hours. (β) Cases with preliminary gastric disturbance, such as nausea and vomiting, or some local affection, as pharyngitis, phlegmon, or a pulmonary complication. In such cases the attack begins with headache, delirium, great distress, and dyspnœa, affecting both inspiration and expiration, a condition called by Küssmaul *air-hunger*. Cyanosis may or may not be present. If it is, the pulse becomes rapid and weak and the patient gradually sinks into coma; the attack lasting from one to five days. There may be a very heavy, sweetish odor of the breath, due to the presence of acetone. (γ) Cases in which, without any previous dyspnœa or distress, the patient is attacked with headache and a feeling of intoxication, and rapidly falls into a deep and fatal coma.

There has been much dispute as to the nature of these symptoms, but our knowledge of the disease is not yet sufficiently advanced to give a rational explanation. The character of the attack and the similarity, in many instances, to uræmia would indicate that it depended upon some toxic agent in the blood. The theory most commonly held, that this material is acetone, is supported by the presence of the acetone reaction in the urine and its odor in the breath. Stadelmann believes that the condition is not acetonæmia, but that the poisonous agent is an intermediate product between the sugar and acetone, an oxy-butyric acid.

Saunders and Hamilton have described cases in which the lung capillaries were blocked with fat. They attributed the symptoms to fat embolism, but there are many cases on record in which this condition was not found, though lipæmia is by no means infrequent in diabetes.

The symptoms have been attributed to uræmia, and albuminuria frequently precedes or accompanies the attack.

(2) *Peripheral Neuritis.*—The *neuralgias*, numbness, and tingling, which are not uncommon symptoms in diabetes, are probably minor neuritic manifestations.

Diabetic Tabes (so called).—This is a peripheral neuritis, characterized by lightning pains in the legs, loss of knee-jerk—which may occur without the other symptoms—and a loss of power in the extensors of the feet. The gait is the characteristic *steppage*, as in arsenical, alcoholic, and other

forms of neuritic paralysis. Charcot states that there may be atrophy of the optic nerves.

Diabetic Paraplegia.—This is also in all probability due to neuritis. There are cases in which power has been lost in both arms and legs.

(3) *Mental Symptoms.*—The patients are often morose, and there is a strong tendency to become hypochondriacal. General paralysis has been known to develop.

(4) *Special Senses.*—Cataract is liable to occur, and may develop with rapidity in young persons. Diabetic retinitis closely resembles the albuminuric form. Hæmorrhages are common. Sudden amaurosis, similar to that which occurs in uræmia, may occur. Paralysis of the muscles of accommodation may be present; and lastly atrophy of the optic nerves. Aural symptoms may come on with great rapidity, either an otitis media, or in some instances inflammation of the mastoid cells.

(5) *Sexual Function.*—Impotence is common, and may be an early symptom.

Course.—In children the disease is rapidly progressive, and may prove fatal in a few days. It may be stated, as a general rule, that the older the patient at the time of onset the slower the course. Cases without hereditary influences are the most favorable. In stout, elderly men diabetes is a much more hopeful disease than it is in thin persons. Middle-aged patients may live for many years, and persons are met with who have had the disease for ten, twelve, or even fifteen years.

Diagnosis.—Glycosuria, which to all intents and purposes is a mild form of the disease, is to be distinguished only by its transient character. There is no other disease with which true diabetes can be confounded. It must not be forgotten that hysterical women sometimes put sugar in the urine for the purposes of deception.

Prognosis.—In true diabetes instances of cure are rare. On the other hand, the transient or intermittent glycosuria, met with in stout overfeeders, or in persons who have undergone a severe mental strain, is very amenable to treatment. Not a few of the cases of reputed cures belong to this division. Personally I have never seen *recovery* from a case of true diabetes. Temporary arrest, reduction to a minimum of the amount of sugar excreted, and prolonged periods of good health, I have frequently seen, but neither in any one of my personal friends or acquaintances who have suffered with the disease, nor in patients who have come under my care in hospital or private practice, have I known permanent and complete disappearance of the sugar, so that an ordinary diet could be taken with impunity. Cures are, however, reported. Practically, in cases under forty years of age the outlook is bad; in older persons the disease is less serious and much more amenable to treatment.

Treatment.—In families with a marked predisposition to the disease the use of starchy and saccharine articles of diet should be restricted.

The personal hygiene of a diabetic patient is of the first importance.

Sources of worry should be avoided, and he should lead an even, quiet life, if possible in an equable climate. Flannel or silk should be worn next to the skin, and the greatest care should be taken to promote its action. A lukewarm, or if tolerably robust, a cold bath, should be taken every day. An occasional Turkish bath is useful. Systematic, moderate exercise should be taken. When this is not feasible, massage should be given.

Diet.—Our injunctions to-day are those of Sydenham: "Let the patient eat food of easy digestion, such as veal, mutton, and the like, and abstain from all sorts of fruit and garden stuff."

The *carbohydrates* in the food should be reduced to a minimum. Under a strict hydrocarbonaceous and nitrogenous regimen all cases are benefited and some are cured. The most minute and specific instructions should be given in each case, and the dietary arranged with scrupulous care. It is of the first importance to give the patient variety in the food, otherwise the loathing of certain essential articles becomes intolerable, and too often the patient gives up in disgust or despair. It is well, perhaps, not to attempt the absolute exclusion of the carbohydrates, but to allow a small proportion of ordinary bread, or, better still, as containing less starch, potatoes. It is best gradually to enforce a rigid system, cutting off one article after another. The following is a list of articles which diabetic patients may take:

Liquids: Soups — ox-tail, turtle, bouillon, and other clear soups. Lemonade, coffee, tea, chocolate, and cocoa; these to be taken without sugar, but they may be sweetened with saccharin. Potash or soda water, and the Apollinaris, or the Saratoga Vichy, and milk in moderation, may be used.

Of animal food: Fish of all sorts, salt and fresh, butcher's meat (with the exception of liver), poultry, and game. Eggs, butter, buttermilk, curds, and cream cheese.

Of bread: Gluten and bran bread, and almond and cocoanut biscuits.

Of vegetables: Lettuce, tomatoes, spinach, chiccory, sorrel, radishes, water-cress, mustard and cress, cucumbers, celery, and endives. Pickles of various sorts.

Fruits: Lemons, oranges, and currants. Nuts are, as a rule, allowable.

Among *prohibited articles* are the following: Thick soups, liver, crabs, lobsters, and oysters; though, if the livers are cut out, oysters may be used.

Ordinary bread of all sorts (in quantity): rye, wheaten, brown, or white. All farinaceous preparations, such as hominy, rice, tapioca, semolina, arrowroot, sago, and vermicelli.

Of vegetables: Potatoes, turnips, parsnips, squashes, vegetable marrow of all kinds, beets, corn, artichokes, and asparagus.

Of liquids: Beer, sparkling wine of all sorts, and the sweet aërated drinks.

The chief difficulty in arranging the daily *menu* of a diabetic patient is the bread, and for it various substitutes have been advised—bran bread, gluten bread, and almond biscuits. Most of these are unpalatable, and the patients weary of them rapidly. Too many of them are gross frauds, and contain a very much greater proportion of starch than represented. A friend, a distinguished physician, who has, unfortunately, had to make trial of a great many of them, writes: "That made from almond flour is usually so heavy and indigestible that it can only be used to a limited extent. Gluten flour obtained in Paris or London contains about 15 per cent of the ordinary amount of starch and can be well used. The gluten flour obtained in this country has from 35 to 45 per cent of starch, and can be used successfully in mild but not in severe forms of diabetes."

Unless a satisfactory and palatable gluten bread can be obtained, it is better to allow the patient a few ounces of ordinary bread daily. The "Soya" bread is not any better than that made from the best gluten flour. As a substitute for sugar, saccharin is very useful, and is perfectly harmless. Glycerin may also be used for this purpose.

It is well to begin the treatment by cutting off article after article until the sugar disappears from the urine. Within a month or two the patient may gradually be allowed a more liberal regimen. An exclusively milk diet, either skimmed milk, buttermilk, or koumyss, has been recommended by Donkin and others. Certain cases seem to improve on it, but it is not, on the whole, to be recommended.

Medicinal Treatment.—This is most unsatisfactory, and no one drug appears to have a directly curative influence. Opium alone stands the test of experience as a remedy capable of limiting the progress of the disease. Diabetic patients seem to have a special tolerance for this drug. Codeia is preferred by Pavy, and has the advantage of being less constipating than morphia. A patient may begin with half a grain three times a day, which may be gradually increased to six or eight grains in the twenty-four hours. Mitchell Bruce, from a series of elaborate observations, concludes that morphia is decidedly more powerful. In a patient at the University Hospital, Philadelphia, on whom I made a large number of observations on the comparative value of these drugs, morphia appeared to be much more potent. Patients take with benefit up to five or six grains in the twenty-four hours. The expense, too, must sometimes be taken into consideration: the cost of six grains of codeia daily would be twenty-five cents, whereas the same amount of morphia would cost only ten cents. Not much effect is noticed unless the patient is on a rigid diet. When the sugar is reduced to a minimum, or is absent, the opium should be gradually withdrawn. The patients not only bear well these large doses of morphia, but they stand its gradual reduction. Potassium bromide is often a useful adjunct. The arsenite of bromine, a solution of arsenious acid with bromine in glycerin (dose, three to five minims after meals), has been very highly recommended, but it is by no means so cer-

tain as opium. Arsenic alone may be used. Antipyrin may be given in doses of ten grains three times a day, and in cases with a marked neurotic constitution is sometimes satisfactory. The salicylates, iodoform, nitroglycerin, jambul, lithium salts, strychnine, creasote, and lactic acid have been employed.

Of the complications, the *pruritis* and *eczema* are best treated by cooling lotions of boric acid or hyposulphite of soda (1 ounce; water, 1 quart).

The *coma* is an almost hopeless complication. Inhalations of oxygen have been recommended, and lately the intravenous injections of a saline solution, as practised by Hilton Fagge. The three per cent solution of the sodium bicarbonate has generally been employed. The treatment has not, however, been satisfactory. Of seventeen cases, collected by Chadbourne, in only one was it successful; in seven there was temporary improvement; and the best that can be said for it is that it may give the patient a few hours of complete consciousness. Injections should be made as soon as possible after the appearance of the coma.

VIII. DIABETES INSIPIDUS.

Definition.—A chronic affection characterized by the passage of large quantities of normal urine of low specific gravity.

The condition is to be distinguished from diuresis or polyuria, which is a frequent symptom in hysteria, in Bright's disease, and occasionally in cerebral or other affections. Willis, in 1674, first recognized the distinction between a saccharine and non-saccharine form of diabetes.

Etiology.—The disease is most common in young persons. Of the 85 cases collected by Strauss, 9 were under five years; 12 between five and ten years; 36 between ten and twenty-five years. Males are more frequently attacked than females. The affection may be congenital. A hereditary tendency has been noted in many cases, the most extraordinary of which has been reported by Weil. Of 91 members in four generations, 23 had persistent polyuria without any deterioration in health. Injury to the nervous system has been present in certain instances, and the disease has followed sunstroke, or a violent emotion, such as fright. Traumatism has occasionally been the exciting cause. The injury may have been to the head, but in other cases the lesion has been to the trunk or to the limbs. The disease has followed rapidly the copious drinking of cold water, or a drinking-bout; or has set in during the convalescence from an acute disease. Tumors of the brain and lesions of the medulla have been met with in a few instances. Cases of polyuria have been accompanied by paralysis of the sixth nerve. Maguire has seen an instance after meningitis in which paralysis of the sixth pair occurred with it. Bernard, it will be remembered, discovered a spot in the floor of the fourth ventricle

of animals which, when punctured, produced polyuria. Lesions of the organs of the abdomen may be associated with an excessive flow of urine, which, however, should not be regarded as true diabetes insipidus. Dickenson mentions its occurrence in abdominal tumors; Ralfe, in abdominal aneurism. I have noted it in several cases of tuberculous peritonitis.

The nature of the disease is unknown. It is, doubtless, of nervous origin. The most reasonable view is that it results from a vaso-motor disturbance of the renal vessels, due either to local irritation, as in a case of abdominal tumor, or to central disturbance in cases of brain-lesion, or to functional irritation of the centre in the medulla, giving rise to continuous renal congestion.

Morbid Anatomy.—There are no constant anatomical lesions. The *kidneys* have been found enlarged and congested. The *bladder* has been found hypertrophied. Dilatation of the ureters and of the pelves of the kidneys has been present. Death has not infrequently resulted from ıronic pulmonary disease. Very varied lesions have been met with in ıe nervous system.

Symptoms.—The disease may come on rapidly, as after a fright or an injury. More commonly it develops slowly. A copious secretion of urine, with increased thirst, are the prominent features of the disease. The amount of urine in the twenty-four hours may range from twenty to forty pints, or even more. The specific gravity is low, 1·001 to 1·005; the color is extremely pale and watery. The total solid constituents may not be reduced. The amount of urea has sometimes been found in excess. Abnormal ingredients are rare. Muscle sugar, inosite, has been occasionally found. Albumen is rare. Traces of sugar have been met with. Naturally, with the passage of such enormous quantities of urine, there is a proportionate thirst, and the only inconvenience of the disease is the necessity for frequent micturition and frequent drinking. The appetite is usually good, rarely excessive as in diabetes mellitus. The patients may be well nourished and healthy-looking. The disease in many instances does not appear to interfere in any way with the general health. The perspiration is naturally slight and the skin is harsh. The amount of saliva is small and the mouth usually dry. Cases have been described in which the tolerance of alcohol has been remarkable, and patients have been known to take a couple of pints of brandy, or a dozen or more bottles of wine, in the day.

The course of the disease depends entirely upon the nature of the primary trouble. Sometimes, with organic disease, either cerebral or abdominal, the general health is much impaired; the patient becomes thin, and rapidly loses strength. In the essential or idiopathic cases, good health may be maintained for an indefinite period, and the affection has been known to persist for fifty years. Death usually results from some intercurrent affection. Spontaneous cure may take place.

Diagnosis.—A low specific gravity and the absence of sugar in the

urine distinguish the disease from diabetes mellitus. Hysterical polyuria may sometimes simulate it very closely. The amount of urine excreted may be enormous, and only the development of other hysterical manifestations may enable the diagnosis to be made. This condition is, however, always transitory.

In certain cases of chronic Bright's disease a very large amount of urine of low specific gravity may be passed, but the presence of albumen and of hyaline casts, and the existence of heightened arterial tension, stiff vessels, and hypertrophied left ventricle make the diagnosis easy.

Treatment.—The treatment is not satisfactory. No attempt should be made to reduce the amount of liquid. Opium is highly recommended, but is of doubtful service. The preparations of valerian may be tried; either the powdered root, beginning with five grains three times a day, and increasing until two drachms are taken in the day, or the valerianate of zinc, in fifteen-grain doses, gradually increased to thirty grains, three times a day. Ergot is recommended by DaCosta. Ergotin may be employed. Large doses are required. Antipyrin, the salicylates, arsenic, strychnine, turpentine, and the bromides have been recommended. The constant current may be used—one pole on the loins, the other on the nape of the neck.

IX. RICKETS.

Definition.—A disease of infants, characterized by impaired nutrition and alterations in the growing bones.

Glisson, the anatomist of the liver, described the disease accurately in the seventeenth century.

Etiology.—The disease exists in all parts of the world, but is particularly marked among the poor of the larger cities, who are badly housed and ill fed. It is much more common in Europe than in America. In the colored race it is frequently seen. It is a comparatively rare disease in Canada. In the larger cities of this continent it is frequently seen at the clinics, but in comparison with Vienna and London the contrast is very striking. In these cities from 50 to 80 per cent of all the children at the clinics present signs of rickets. Want of sunlight and impure air are important factors. A starchy diet, too much cows' milk, and the indiscriminate feeding, so common in the children of the poor, are important agents; but something is required beyond these, for children of healthy parents, who have an ample quantity of the proper food, may become rickety. It seems probable, however, that the combination of defective food and bad air plays the most important *rôle*. Prolonged lactation or suckling a child during pregnancy are accessory etiological factors.

There is no evidence that the disease is hereditary, but there is probably a form of fœtal rickets. It is doubtful, however, whether the changes met with in this are identical with the post-natal disease. In these babies,

which are generally still-born, the limbs are short, the curves of the bones are exaggerated, and at the junction of the epiphyses there is no proliferating zone of cartilage. This condition, which Parrot calls *achondroplasy*, is really more like a fœtal *cretinism*.

Rickets affects male and female children equally. It is a disease of the first and second years of life, rarely beginning before the sixth month. Jenner has described a late rickets, in which form the disease may not appear until the ninth or even until the twelfth year. It has been held that rickets is only a manifestation of congenital syphilis (Parrot), but this is certainly not correct. Syphilitic bones rarely, if ever, present the spongy tissue peculiar to rickets, and rachitic bones never show the multiple osteophytes of syphilis. It has been regarded as an effect of malaria.

Morbid Anatomy.—The bones show the most important changes, particularly the ends of the long bones and the ribs. Between the shaft and epiphyses a slight bulging is apparent, and on section the zone of proliferation, which normally is represented by two narrow bands, is greatly thickened, bluish in color, more irregular in outline, and very much softer. The width of this cushion of cartilage varies from five to fifteen millimetres. The line of ossification is also irregular and more spongy and vascular than normal. The periosteum strips off very readily from the shaft, and beneath it there may be a spongioid tissue not unlike decalcified bone. The practical outcome of these changes is a delay in, and imperfect performance of, the ossification, so that the bone has neither the natural rate of growth nor the normal firmness. In the cranium there may be large areas, particularly in the parieto-occipital region, in which the ossification is delayed, producing the so-called cranio-tabes, so that the bone yields readily to pressure with the finger. There are localized depressed spots of atrophy, which, on pressure, give the so-called "parchment crackling." Flat hyperostoses develop from the outer table, particularly on the frontal and parietal bones, and produce the characteristic broad forehead with prominent frontal eminences, a condition sometimes mistaken for hydrocephalus.

The chemical analysis of rickety bones shows a marked diminution in the calcareous salts, which may be as low as from 25 to 35 per cent.

The liver and spleen are usually enlarged, and sometimes the mesenteric glands. As Gee suggests, these conditions probably result from the general state of the health associated with rickets. It is interesting to note that Beneke describes a relative increase in the size of the arteries in rickets.

Kassowitz, who may be considered the leading authority on the anatomy of rickets, regards the hyperæmia of the periosteum, the marrow, the cartilage, and of the bone itself as the primary lesion, out of which all the others develop. This disturbs the normal development of the growing bone, and excites changes in the bone already formed. The cartilage cells in consequence proliferate, the matrix is softer, and the bone which is

formed from this unhealthy cartilage is lacking in firmness and solidity. In the bone already formed this excessive vascularity favors the normal processes of absorption, so that the relation between removal and deposition is disturbed, absorption taking place more rapidly. The new material is poor in lime salts. Kassowitz seems to have proved experimentally that hyperæmia of bone results in defective deposition of lime salts. Barlow and Bury * have given an elaborate analysis of the changes described by this author. It is interesting to note that Glisson attributed rickets to disturbed nutrition by arterial blood, and believed the changes in the long bones to be due to excessive vascularity.

Symptoms.—The disease comes on insidiously about the period of dentition, before the child begins to walk. In many cases digestive disturbances precede the appearance of the characteristic lesions, and the nutrition of the child is markedly impaired. There is usually slight fever, the child is irritable and restless, and sleeps badly. If the child has already walked, it shows a marked disinclination to do so, and seems feeble and unsteady in its gait. Sir William Jenner has called attention to three general symptoms which are present in many cases of rickets. There is first a diffuse soreness of the body, so that the child cries when an attempt is made to move it, and prefers to keep perfectly still. This tenderness is often a marked and suggestive symptom. Associated with this are slight fever and a tendency at night to throw off the bedclothes. This may be partly due to the fact that the general sensitiveness is such that even their weight may be distressing. And, third, there is such profuse sweating, particularly about the head and neck, that in the morning the pillow is found soaked with perspiration.

The tissues become soft and flabby; the skin is pale; and from a healthy, plump condition, the child becomes puny and feeble. It is in this stage of the disease that we sometimes find such a degree of disability in the muscles, particularly of the legs, that paralysis may be suspected. This so-called pseudo-paresis of rickets results in part from the flabby, weak condition of the legs and in part from the pain associated with the movements. Such cases are by no means uncommon, but they are readily distinguished from infantile paralysis. Coincident with, or following closely upon, the general symptoms the characteristic skeletal lesions are observed. Among the first of these to appear are the changes in the ribs, at the junction of the bone with the cartilage, forming the so-called rickety rosary. When the child is thin these nodules may be distinctly seen, and in any case can be easily made out by touch. They very rarely appear before the third month. They may increase in size up to the second year, and are rarely seen after the fifth year. The thorax undergoes important changes. Just outside the junction of the cartilages with the ribs there is an oblique, shallow depression extending downward and out-

* Cyclopædia of the Diseases of Children, vol. ii.

ward. A transverse curve, sometimes called Harrison's groove, passes outward from the level of the ensiform cartilage toward the axilla and may be deepened at each inspiration. It is rendered more prominent by the eversion and prominence of the costal border. The sternum projects, particularly in its lower half, forming the so-called pigeon or chicken breast. These changes in the thorax are not peculiar, however, to rickets, and are much more commonly associated with hypertrophy of the tonsils, or any trouble which interferes with the free entrance of air into the lungs. Posteriorly the spine is usually curved, the processes are prominent, and lateral curvature may be produced.

The head of a rickety child usually looks large, and the fontanelles remain open for a long time. There are areas, particularly in the parieto-occipital regions, in which ossification is imperfect; and the bone may yield to the pressure of the finger, a condition to which the term *cranio-tabes* has been given. The relation of this condition to rickets is still somewhat doubtful, as it is very often associated with syphilis—in 47 of 100 cases recently studied by George Carpenter. Coincidently with this, hyperplasia proceeds in the frontal and parietal eminences, so that these portions of the skull increase in thickness, and may form irregular bosses. In one type the skull may be large and elongated, with the top considerably flattened. In another, and perhaps more common case, the shape of the skull, when seen from above, is rectangular—the *caput quadratum.* The skull looks large in proportion to the face. The forehead is broad and square, and the frontal eminences marked. The anterior fontanelle is late in closing and may remain open until the third or fourth year. The skin is thin, the veins are perceptible, and the hair is often rubbed from the back of the skull. In contradistinction to the cranio-tabes is the condition of cranio-sclerosis, which has also been ascribed to rickets.

On placing the ear over the anterior fontanelle, or in the temporal region, a systolic murmur may frequently be heard. This condition, first described by Fisher, of Boston, was believed by him to be peculiar to rickets. While unquestionably heard with the greatest frequency in this disease, its presence and persistence in perfectly healthy infants have been amply demonstrated.* The murmur is rarely heard after the fifth year. A knowledge of the existence of this systolic brain murmur may prevent errors. A case in which it was well marked was reported as an instance of supposed gummy tumor of the brain, in which the murmur was thought to be due to pressure on the vessels at the base.

Changes occur in the bones of the face, chiefly in the maxillæ, which are reduced in size. The normal process of dentition is much disturbed; indeed, late teething is one of the marked features in rickets. The teeth which appear may be small and badly formed.

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* Osler, On the Systolic Brain Murmur of Children, Boston Medical and Surgical Journal, 1880.

In the upper limbs changes in the scapulæ are not common. The clavicle may be thickened at the sternal end, and there may be thickening near the attachment of the sterno-cleido muscle. The most noticeable changes are at the lower ends of the radius and ulna. The enlargement is at the junction-area of the shaft and epiphysis. Less evident enlargements may occur at the lower end of the humerus. In severe cases the natural shape of the bones of the arm may be much altered, having to support the weight of the child in crawling on the floor. The changes in the pelvis are of special importance, particularly in female children, as in extreme cases they lead to great deformity and narrowing of the outlet. In the legs, the lower end of the tibia first becomes enlarged; and in slight cases it may alone be affected. In the severe forms the upper end of the bone, the corresponding parts of the fibula, and the lower end of the femur become greatly thickened. If the child walks, slight bowing of the tibiæ inevitably results. In more advanced cases the tibiæ and even the femora may be arched forward. In other cases the condition of knock-knee occurs. Unquestionably the chief cause of these deformities is the weight of the body in walking, but muscular action takes part in it. The green-stick fracture is not uncommon in the soft bones of rickets.

These changes in the skeleton proceed slowly, and the general symptoms vary a good deal with their progress. The child becomes more or less emaciated, though "fat rickets" is by no means uncommon. Fever is not constant, but in actively progressing changes in the bone there is usually a slight pyrexia. The abdomen is large, due partly to flatulent distention, partly to enlargement of the liver, and in severe cases to diminution of the volume of the thorax. The spleen is often enlarged and readily palpable. The urine is stated to contain an excess of lime salts, but Jacobi and Barlow say this has not been proved. No special or peculiar changes, indeed, have as yet been described. Many rickety children show marked nervous symptoms; irritability, peevishness, and sleeplessness are constantly present. Jenner called attention to the close relationship which existed between rickets and infantile convulsions, particularly to the fits which occur after the sixth month. Tetany is by no means uncommon. It involves most frequently the arms and hands; occasionally the legs as well. Laryngismus stridulus is a common complication, and though not, as some state, invariably associated with this disease, yet it is certainly much more frequent in rickety than in other children. Severe rickets interfere seriously with the growth of a child. Extreme examples of rickety dwarfs are not uncommon. The disease known as acute rickets is in reality a manifestation of scurvy and will be described with that disease.

Prognosis.—The disease is never in itself fatal, but the condition of the child is such that it is readily carried off by intercurrent affections, particularly those of the respiratory organs. Spasm of the larynx and

convulsions occasionally cause death. In females the deformity of the pelvis is serious, as it may lead to difficulties in parturition.

Treatment.—The better the condition of the mother during pregnancy the less likelihood is there of the development of rickets in the child. Rapidly repeated pregnancies and suckling a child during pregnancy seem important factors in the production of the disease. Of the general treatment, attention to the feeding of the child is the first consideration. If the mother is unhealthy, or cannot from any cause nurse the child, a suitable wet-nurse should be provided, or the child must be artificially fed. Cows' milk, diluted according to the age of the child, should constitute the chief food. Care should be taken to examine the condition of the stools, and if curds are present the child is taking too much, or it is not sufficiently diluted. Barley-water or carefully strained and well-boiled oatmeal gruel form excellent additions to the milk.

The child should be warmly clad and should be in the fresh air and sunshine the greater part of the day. It is a "vulgar error" to suppose that delicate children cannot stand, when carefully wrapped up, an even low temperature. The child should be bathed daily in warm water. Careful friction with sweet oil is very advantageous, and, if properly performed, allays rather than aggravates the sensitiveness. Special care should be taken to prevent deformity. The child should not be allowed to walk, and for this purpose splints applied so as to extend beyond the feet are very effective. Of medicines, phosphorus has been warmly recommended by Kassowitz, and its use is also advised by Jacobi. The child may be given gr. $\frac{1}{120}$ two or three times a day, dissolved in olive oil. Cod-liver oil, in doses of from a half to one teaspoonful, is very advantageous. The syrup of the iodide of iron may be given with the oil. The digestive disturbances, together with the respiratory and nervous complications, should receive appropriate treatment.

X. SCURVY (*Scorbutus*).

Definition.—A constitutional disease characterized by great debility, with anæmia, a spongy condition of the gums, and a tendency to hæmorrhages.

Etiology.—The disease has been known from the earliest times, and has prevailed particularly in armies in the field and among sailors on long voyages.

From the early part of this century, owing largely to the efforts of Lind and to a knowledge of the conditions upon which the disease depends, scurvy has gradually disappeared from the naval service. In the mercantile marine, cases still occasionally occur, owing to neglect of proper and suitable food.

The disease develops whenever individuals have subsisted for pro-

longed periods upon a diet in which fresh vegetables or their substitutes are lacking.

In comparison with former times it is now a rare disease. In seaport towns sailors suffering with the disease are occasionally admitted to hospitals. In large almshouses, during the winter, cases are occasionally seen.* On several occasions in Philadelphia characteristic examples were admitted to my wards from the almshouse. Some years ago it was not very uncommon among the lumbermen in the winter camps in the Ottawa Valley. Among the Hungarian, Bohemian, and Italian miners in Pennsylvania, cases of the disease are not infrequent. This so-called land scurvy differs in no particular from the disease in sailors. An insufficient diet appears to be an essential element in the disease, and all observers are now unanimous that it is the absence of those ingredients in the food which are supplied by fresh vegetables. What these constituents are has not yet been definitely determined. Garrod holds that the defect is in the absence of the potassic salts. Others believe that the essential factor is the absence of the organic salts present in fruits and vegetables. Ralfe, who has made a very careful study of the subject, believes that the absence from the food of the malates, citrates, and lactates reduces the alkalinity of the blood, which depends upon the carbonates directly derived from these salts. This diminished alkalinity, gradually produced in the scurvy patients, is, he believes, identical with the effect which can be artificially produced in animals by feeding them with an excess of acid salts; the nutrition is impaired, there are ecchymoses, and profound alterations in the characters of the blood. The acidity of the urine is greatly reduced and the alkaline phosphates are diminished in amount.

In opposition to this chemical view it has been urged that the disease really depends upon a specific micro-organism.

Other factors play an important part in the disease, particularly physical and moral influences; overcrowding, dwelling in cold, damp quarters, and prolonged fatigue under depressing influences, as during the retreat of an army. Among prisoners, mental depression plays an important *rôle*. It is stated that epidemics of the disease have broken out in the French convict-ships *en route* to New Caledonia, even when the diet was amply sufficient. Nostalgia is sometimes an important element. It is an interesting fact that prolonged starvation in itself does not necessarily cause scurvy. Not one of the professional fasters of late years has displayed any scorbutic symptom. The disease attacks all ages, but the old are more susceptible to it. Sex has no special influence, but during the siege of Paris it was noted that the males attacked were greatly in excess of the females. Infantile scurvy will be considered in a special note.

Morbid Anatomy.—The anatomical changes are marked, though

* Henry, Philadelphia Hospital Reports, vol. i, 1890.

by no means specific, and are chiefly those associated with hæmorrhage. The blood is dark and fluid. There are no characteristic microscopical alterations. The bacteriological examination has not yielded anything very positive. Practically there are no changes in the blood, either anatomical or chemical, which can be regarded as peculiar to the disease. The skin shows the ecchymoses evident during life. There are hæmorrhages into the muscles, and occasionally about or even into the joints. Hæmorrhages occur in the internal organs, particularly on the serous membranes and in the kidneys and bladder. The gums are swollen and sometimes ulcerated, so that in advanced cases the teeth are loose, and have even fallen out. Ulcers are occasionally met with in the ileum and colon. Hæmorrhages are extremely common into the mucous membranes. The spleen is enlarged and soft. Parenchymatous changes are constant in the liver, kidneys, and heart.

Symptoms.—The disease is insidious in its onset. Early symptoms are loss in weight, progressively developing weakness, and pallor. Very soon the gums are noticed to be swollen and spongy, to bleed easily, and in extreme cases to present a fungous appearance. The teeth may become loose and even fall out. Actual necrosis of the jaw is not common. The breath is excessively foul. The tongue is swollen, but may be red and not much furred. The salivary glands are occasionally enlarged. The lesions of the gums are rarely absent. The skin becomes dry and rough, and ecchymoses soon appear, first on the legs and then on the arms and trunk. They are petechial, but may become larger, and when subcutaneous may cause distinct swellings. In severe cases, particularly in the legs, there may be effusion between the periosteum and the bone, forming irregular nodes, which, in the case of a sailor from a whaling vessel, who came under my observation, had broken down and formed foul-looking sores. The slightest bruise or injury causes hæmorrhage into the injured part. Œdema about the ankles is common. Hæmorrhages from the mucous membranes are less constant symptoms. Epistaxis is, however, frequent. Hæmoptysis and hæmatemesis are uncommon. Hæmaturia and bleeding from the bowels may be present in very severe cases.

Palpitation of the heart and feebleness and irregularity of the impulse are prominent symptoms. A hæmic murmur can usually be heard at the base. Hæmorrhagic infarction of the lungs and spleen has been described. Respiratory symptoms are not common. The appetite is impaired, and owing to the soreness of the gums the patient is unable to chew the food. Constipation is more frequent than diarrhœa. The urine is often albuminous. The changes in the composition of the urine are not constant; the specific gravity is high; the color is deeper; and the phosphates are increased. The statements with reference to the inorganic constituents are contradictory. Some say the phosphates and potash are deficient; others that they are increased.

There are mental depression, indifference, in some cases headache, and

in the latter stages delirium. Cases of convulsions, of hemiplegia, and of meningeal hæmorrhage have been described. Remarkable ocular symptoms are occasionally met with, such as night-blindness or day-blindness.

In advanced cases necrosis of the bones may occur, and in young persons even separation of the epiphyses. There are instances in which the cartilages have separated from the sternum. The callus of a recently repaired fracture has been known to undergo destruction. Fever is not present, except in the later stages, or when secondary inflammations in the internal organs appear. The temperature may indeed be sometimes below normal.

Scurvy in Children.—In infants and young children, fed upon improper food, a form of cachexia develops which has been regarded as acute rickets, but which Cheadle and Barlow have shown to be a form of scurvy. The most striking cases develop in infants reared on artificial food prepared with water, though the disease has occurred when these foods were prepared with milk. Rickets strongly predispose to the condition. The cases may occur in infants, or in children up to the age of ten. Barlow thus summarizes the chief features:

"(1) Predominance of lower limb affection:

"(*a*) Immobility, going on to pseudo-paralysis; (*b*) excessive tenderness; (*c*) general swelling of lower limbs; (*d*) skin shiny and tense, but seldom pitting, and not characterized by undue local heat; (*e*) on subsidence, revealing a deep thickening of the shaft; (*f*) liability to fracture near the epiphyses.

"(2) Swelling of the gums, varying from definite sponginess down to a vanishing-point of minute transient ecchymoses. These constitute the chief diagnostic differentia between infantile scurvy and rickets, properly so called. But to them must be added, as the most important diagnostic of all, (3) definite and rapid amelioration by antiscorbutic regimen." According to Gee, hæmaturia may be the only sign of scurvy in children.

Diagnosis.—No difficulty is met in the recognition of scurvy when a number of persons are affected together. In isolated cases, however, the disease is distinguished with difficulty from certain forms of purpura. The association with manifest insufficiency in diet, and the rapid amelioration with suitable food, are points by which the diagnosis can be readily settled.

Prognosis.—The outlook is good, unless the disease is far advanced and the conditions persist which lead to its development. The mortality now is rarely great. During the civil war the death-rate was sixteen per cent. Death results from gradual heart-failure, occasionally from sudden syncope. Meningeal hæmorrhage, extravasation into the serous cavities, entero-colitis, and other intercurrent affections may prove fatal.

Prophylaxis.—The regulations of the Board of Trade require that a sufficient supply of antiscorbutic articles of diet is taken on each ship; so that now, except as the result of accident, the occurrence of scurvy on

board a vessel should lead to the indictment of the captain or owners for criminal negligence. An outbreak of the disease in an almshouse is evidence of culpable neglect on the part of the managers.

Treatment.—The juice of two or three lemons daily and a varied diet, with plenty of fresh vegetables, suffice to cure all cases of scurvy, unless far advanced. When the stomach is much disordered, small quantities of scraped meat and milk should be given at short intervals, and the lemon-juice in gradually increasing quantities. A bitter tonic, or a steel and bark mixture, may be given. As the patient gains in strength, the diet may be more liberal and he may eat freely of potatoes, cabbage, water-cresses, and lettuce. The stomatitis is the symptom which causes the greatest distress. The permanganate of potash or dilute carbolic acid forms the best mouth-wash. Pencilling the swollen gums with a tolerably strong solution of nitrate of silver is very useful. The solution is better than the solid stick, as it reaches to the crevices between the granulations. The constipation which is so common is best treated with large enemata. For other conditions, such as hæmorrhages and ulcerations, suitable measures must be employed.

XI. PURPURA.

Strictly speaking this is a symptom, not a disease; but under this term are conveniently arranged a number of affections characterized by extravasations of the blood into the skin. The purpuric spots vary from one to three or four millimetres in diameter. When small and pin-point-like they are called petechiæ; when large, they are known as ecchymoses. At first bright red in color, they become darker, and gradually fade to brownish stains. They do not disappear on pressure.

It is extremely difficult to make a satisfactory classification of purpura. Perhaps as good a division as can be made is the following:

Symptomatic Purpura.—(*a*) **Infectious.**—In pyæmia, septicæmia, malignant endocarditis (particularly in the latter affection), ecchymoses may be very abundant. In typhus fever the rash is always purpuric. Measles, scarlet fever, and more particularly small-pox, have each a variety characterized by an extensive purpuric rash.

(*b*) **Toxic.**—The virus of snakes produces with great rapidity extravasation of blood; a condition which has been very carefully studied by Weir Mitchell. Certain medicines, particularly copaiba, quinine, belladonna, mercury, ergot, and the iodides occasionally, are followed by a petechial rash. Under this division, too, comes the purpura associated with jaundice.

(*c*) **Cachectic.**—Under this heading are best described the instances of purpura which develop in the constitutional disturbance of cancer, tuberculosis, Hodgkin's disease, Bright's disease, scurvy, and in the debility of

old age. In these cases the spots are usually confined to the extremities. They may be very abundant in the lower limbs and about the wrists and hands. This constitutes, probably, the commonest variety of the disease, and many examples of it can be seen in the wards of any large hospital.

(*d*) **Neurotic.**—One variety is met with in cases of organic disease. It is the so-called myelopathic purpura, which is seen occasionally in locomotor ataxia, particularly following attacks of the lightning pains and, as a rule, involving the area of the skin in which the pains have been most intense. Cases have been met with also in acute myelitis and in transverse myelitis, and occasionally in severe neuralgia. Another form is the remarkable hysterical condition in which stigmata, or bleeding points, appear upon the skin.

(*e*) **Mechanical.**—This variety is most frequently seen in venous stasis of any form, as in the paroxysms of whooping-cough and in epilepsy.

Arthritic.—This form is characterized by involvement of the joints. It is usually known, therefore, as rheumatic, though in reality the evidence upon which this view is based is not conclusive. For the present it seems more satisfactory to use the designation arthritic. Three groups of cases may be recognized:

(*a*) A mild form, often known as **Purpura simplex,** seen most commonly in children, in whom, with or without articular pain, a crop of purpuric spots appears upon the legs, less commonly upon the trunk and arms. As pointed out by Graves, this form is not infrequently associated with diarrhœa. The disease is seldom severe. There may be loss of appetite, and slight anæmia. Fever is not, as a rule, present, and the patients get well in a week or ten days. These cases are usually regarded as rheumatic, and are certainly associated, in some instances, with undoubted rheumatic manifestations; yet in a majority of the patients which I have seen the arthritis was slighter than in the ordinary rheumatism of children, and no other manifestations were present.

(*b*) **Peliosis Rheumatica** (Schönlein's Disease).—This remarkable affection is characterized by multiple arthritis, and an eruption which varies greatly in characters, sometimes *purpuric*, more commonly associated with *urticaria* or with *erythema exudativum*. The disease is most common in males between the ages of twenty and thirty. It not infrequently sets in with sore throat, a fever from 101° to 103°, and articular pains. The purpuric rash makes its appearance first on the legs or about the affected joints. It may be a simple purpura or ordinary urticarial wheals. In other instances there are nodular infiltrations, not to be distinguished from erythema nodosum. The combination of wheals and purpura, the *purpura urticans*, is very distinctive. Much more rarely vesication is met with, the so-called *pemphigoid purpura*. The amount of œdema is variable; occasionally it is excessive. In one case, which I saw in Montreal with Molson, the chin and lower lip were enormously swollen, tense, glazed, and deeply ecchymotic. The eyelids were swollen and purpuric,

while scattered over the cheeks and about the joints were numerous spots of purpura urticans. These are the cases which have been described as *febrile purpuric œdema.* The temperature range, in mild cases, is not high, but may reach 102° or 103°.

The urine is sometimes reduced in amount and may be albuminous. The joint affections are usually slight, though associated with much pain, particularly as the rash comes out. Relapses may occur and the disease may return at the same time for several years in succession.

The diagnosis of Schönlein's disease offers no difficulty. The association of multiple arthritis with purpura and urticaria is very characteristic. In a case which I saw with Musser there was endo-pericarditis, and the question at first arose whether the patient had malignant endocarditis with extensive cutaneous infarcts.

Schönlein's peliosis is thought by most writers to be of rheumatic origin, and certainly many of the cases have the characters of ordinary rheumatic fever, *plus* purpura. By many, however, it is regarded as a special affection, of which the arthritis is a manifestation analogous to that which occurs in hæmophilia. The frequency with which sore throat precedes the attack, and the occasional occurrence of endocarditis or pericarditis, are certainly very suggestive of true rheumatism.

The cases usually do well, and a fatal event is extremely rare. The throat symptoms may persist and give trouble. In two instances I have seen necrosis and sloughing of a portion of the uvula.

(*c*) There is an arthritic purpura which presents marked gastro-intestinal and renal symptoms. This not uncommon but little recognized form is met with most frequently in children and sets in usually with pains, but rarely much swelling in the joints. Purpura or purpura urticans develops about them, and the case at first looks like one of so-called rheumatic purpura. Soon other symptoms develop: the child has attacks of severe colic with vomiting and diarrhœa, true gastro-intestinal crises; which may recur with great frequency, particularly at night. There may be hæmorrhage from the bowels and soon renal symptoms. There are albumen and tube-casts, often blood, and sometimes all the symptoms of an intense hæmorrhagic nephritis. The cases may drag on for months. Death may occur from the nephritis, or from the severe gastro-intestinal disturbance. Couty, who has given the best description of this affection, regards it as a form of nervous purpura. This form has an interesting connection with the angio-neurotic œdema, which is also characterized by severe gastro-intestinal crises. Of four cases which have been under my care one died of the nephritis.*

Purpura Hæmorrhagica.—Under this heading may be considered the cases of very severe purpura with hæmorrhages from the mucous membranes. The affection, known as the *morbus maculosus* of Werlhof,

* New York Medical Journal, 1889.

is most commonly met with in young and delicate individuals, particularly in girls; but cases are described in which the disease has attacked

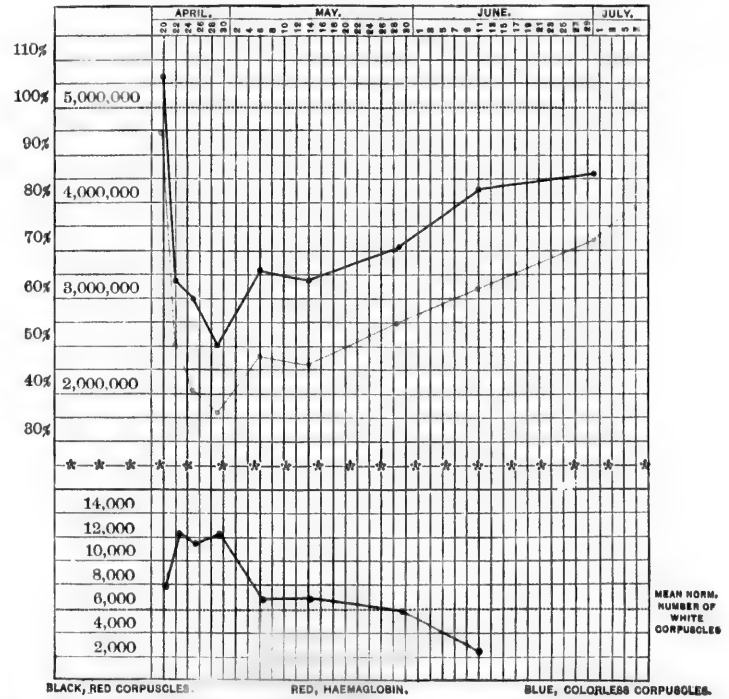


CHART XIV.—Illustrates the rapidity with which anæmia is produced in purpura hæmorrhagica and the gradual recovery.

adults in full vigor. After a few days of weakness and debility, purpuric spots appear on the skin and rapidly increase in numbers and size. Bleeding from the mucous surfaces sets in, and the epistaxis, hæmaturia, and hæmoptysis may cause profound anæmia. Chart XIV illustrates the rapidity with which anæmia is produced and the gradual recovery. Death may take place from loss of blood, or from hæmorrhage into the brain. Slight fever usually accompanies the disease. In favorable cases the affection terminates in from ten days to two weeks. There are instances of purpura hæmorrhagica of great malignancy, which may prove fatal within twenty-four hours—*purpura fulminans*. This form is most commonly met with in children, and is characterized by cutaneous hæmorrhages, which develop with great rapidity. Death may occur before any bleeding takes place from the mucous membranes.

In the *diagnosis* of purpura hæmorrhagica it is important to exclude scurvy, which may be done by the consideration of the previous health,

the circumstances under which the disease develops, and by the absence of swelling of the gums. The malignant forms of the fevers, particularly small-pox and measles, are distingished by the prodromata and the higher temperature.

Treatment.—In symptomatic purpura attention should be paid to the conditions under which it develops, and measures should be employed to increase the strength and to restore a normal blood condition. Tonics, good food, and fresh air meet these indications. In the simple purpura of children, or that associated with slight articular trouble, arsenic in full doses should be given. No good is obtained from the small doses, but the Fowler's solution should be pushed freely until physiological effects are obtained. In peliosis rheumatica the sodium salicylates may be given, but with discretion. I confess not to have seen any special control of the hæmorrhages by this remedy. We are still without a trustworthy medicine which can always be relied upon to control purpura.

Aromatic sulphuric acid, ergot, turpentine, acetate of lead, or tannic and gallic acids, may be used, and in some instances they seem to check the bleeding. In other cases the whole series of hæmostatics may be tried in succession without any benefit.

XII. HÆMOPHILIA.

Definition.—An hereditary, constitutional fault, characterized by a tendency to uncontrollable bleeding, either spontaneous or from slight wounds. It is sometimes associated with a form of arthritis.

Early in the century several physicians of this country called attention to the occurrence of profuse hæmorrhage from slight causes. The fact that fatal hæmorrhage might occur from slight, trifling wounds had been known for centuries. The recognition of the family nature of the disease is due to the writings of Buel, Otto, Hay, Coates, and others in this country. The disease has been elaborately treated in the monographs of Legg and Grandidier.

Etiology.—In a majority of cases the disposition is hereditary. The fault may be acquired, however, but nothing is known of the conditions under which the disease may thus arise in healthy stock.

The hereditary transmission in this disease is remarkable. In the Appleton-Swain family, of Reading, Mass., there have been cases for nearly two centuries; and F. F. Brown, of that town, tells me that instances have already occurred in the seventh generation. The usual mode of transmission is through the mother, who is not herself a bleeder, but the daughter of one. Atavism through the female alone is almost the rule, and the daughters of a bleeder, though healthy and free from any tendency, are almost certain to transmit the disposition to the male offspring. The affection is much more common in males than in females,

the proportion being estimated at eleven to one, or even thirteen to one. The tendency usually appears within the first two years of life. It is rare for manifestations to be delayed until the tenth or twelfth year. Families in all conditions of life are affected. The bleeder families are usually large. The members are healthy-looking, and usually have fine, soft skins.

Morbid Anatomy.—No special peculiarities have been described. In some instances changes have been found in the smaller vessels; but in others careful studies have been negative. An unusual thinness of the vessels has been noted. Hæmorrhages have been found in and about the capsules of the joints, and in a few instances inflammation of the synovial surfaces. The nature of the disease is undetermined, and we do not yet know whether it depends upon a peculiar frailty of the blood-vessels or some peculiarity in the constitution of the blood, which prevents the normal thrombus formation in a wound.

Symptoms.—Usually hæmophilia is not noted in the child until a trifling cut is followed by serious or uncontrollable hæmorrhage, or spontaneous bleeding occurs and presents insuperable difficulties in its arrest. The symptoms may be grouped under three divisions: external bleedings, spontaneous and traumatic; interstitial bleedings, petechiæ and ecchymoses; and the joint affections. The external bleedings may be spontaneous, but more commonly they follow cuts and wounds. In 334 cases (Grandidier) the chief bleedings were epistaxis, 169; from the mouth, 43; stomach, 15; bowels, 36; urethra, 16; lungs, 17; and in a few instances bleeding from the skin of the head, the tongue, finger-tips, tear-papilla, eyelids, external ear, vulva, navel, and scrotum.

Traumatic bleeding may result from blows, cuts, scratches, etc., and the blood may be diffused into the tissues or discharged externally. Trivial operations have proved fatal, such as the extraction of teeth, circumcision, or venesection. It is possible that there may be local defects which make bleeding from certain parts of the body more dangerous. D. Hayes Agnew mentioned to me the case of a bleeder who had always bled from cuts and bruises above the neck, never from those below. The bleeding is a capillary oozing. It may last for hours, or even many days. Epistaxis may prove fatal in twenty-four hours. In the slow bleeding from the mucous surfaces large blood tumors may form and project from the nose or mouth, forming remarkable-looking structures, and showing that the blood has the power of coagulation. The interstitial hæmorrhages may be spontaneous, or may result from injury. Petechiæ or large extravasations—hæmatomata—may occur, the latter usually following blows.

The joint affections of hæmophilia are remarkable. There may simply be pain, or attacks which come on suddenly with fever, and closely resemble acute rheumatism. The larger joints are usually affected. Arthritis may usher in an attack of hæmorrhage.

So far as the examination of the blood goes, no changes of special moment have been noted. When the bleeding has been severe it is thin

and watery, but at the beginning of the bleeding the blood is rich in corpuscles and coagulates firmly.

Diagnosis.—In the diagnosis of the condition the family tendency is important. A single uncontrollable hæmorrhage in child or adult is not to be ranked as hæmophilia; but it is only when a person shows a marked tendency to multiple hæmorrhages, spontaneous or traumatic, which tendency is not transitory but persists, particularly if there have been joint affections, that we may consider the condition hæmophilia. Peliosis rheumatica is an affection which touches hæmophilia very closely, particularly in the relation of the joint swelling. It may also show itself in several members of a family. The diagnosis from the various forms of purpura is usually easy.

Prognosis.—The patients rarely die in the first bleeding. The younger the individual the worse is the outlook, though it is rarely fatal in the first year. Grandidier states that of 152 boy subjects, 81 died before the termination of the seventh year. The longer the bleeder survives the greater the chance of his outliving the tendency; but it may persist to old age, as shown in the case of Oliver Appleton, the first reported American bleeder, who died at an advanced age of hæmorrhage from a bed-sore and from the urethra. The prognosis is graver in a boy than in a girl. In the latter menstruation is sometimes early and excessive, but fortunately, in the female members of hæmophilic families, neither this function nor the act of parturition brings with it special dangers.

Treatment.—Members of a bleeder's family, particularly the boys, should be guarded from injury, and operations of all sorts should be avoided. The daughters should not marry, as it is through them that the tendency is propagated.

When an injury or wound has occurred, absolute rest and compression should first be tried, and if these fail the styptics may be used. In epistaxis ice, tannin, and gallic acid may be tried before resorting to plugging. Internally ergot seems to have done good in several cases. Legg advises the perchloride of iron in half-drachm doses every two hours with a purge of sulphate of soda. Venesection has been tried in several cases. Transfusion has been employed, but without success. During convalescence, iron and arsenic should be freely used.

SECTION III.

DISEASES OF THE DIGESTIVE SYSTEM.

I. DISEASES OF THE MOUTH.

STOMATITIS.

(1) **Acute Stomatitis.**—Simple or erythematous stomatitis, the commonest form of inflammation of the mouth, results from the action of irritants of various sorts. It is frequent at all ages. In children it is often associated with dentition and with gastro-intestinal disturbance, particularly in ill-nourished, unhealthy subjects. In adults it follows the overuse of tobacco and the use of too hot or too highly seasoned food. It is a frequent concomitant of indigestion, and is met with in the acute specific fevers.

The affection may be limited to the gums and lips or may extend over the whole surface of the mouth and include the tongue. There is at first superficial redness and dryness of the membrane, followed by increased secretion and swelling of the tongue, which is furred, and indented by the teeth. There is rarely any constitutional disturbance, but in children there may be slight elevation of temperature. The condition is sufficient to cause considerable discomfort, sometimes amounting to actual distress and pain, particularly in mastication.

In infants the mouth should be carefully sponged after each feeding. A mouth-wash of borax or the glycerine of borax may be used, and in severe cases, which tend to become chronic, a dilute solution of nitrate of silver (three or four grains to the ounce) may be applied.

(2) **Aphthous Stomatitis.**—This form, also known as *follicular* or *vesicular* stomatitis, is characterized by the presence of small, slightly raised spots, from two to four millimetres in diameter, surrounded by reddened areolæ. The spots appear first as vesicles, which rupture, leaving small ulcers with grayish bases and bright-red margins. They are seen most frequently on the inner surfaces of the lips, the edges of the tongue, and the cheeks. They are seldom present on the mucous membrane of the pharynx. This form is met with most often in children under three years. It may occur either as an independent affection or in association with any

one of the febrile diseases of childhood or with an attack of indigestion. The crop of vesicles comes out with great rapidity and the little ulcers may be fully formed within twenty-four hours. The child complains of soreness of the mouth and takes food with reluctance. The buccal secretions are increased, and the breath is heavy, but not foul. The constitutional symptoms are usually those of the disease with which the aphthæ are associated. The disease must not be confounded with thrush. No special parasite has been found in connection with it. It is not a serious condition, and heals rapidly with the improvement of the constitutional state. In severe cases it may extend to the pillars of the fauces and to the pharynx, and produce ulcers which are irritating and difficult to heal.

Each ulcer should be touched with nitrate of silver and the mouth should be thoroughly cleansed after taking food. A wash of chlorate of potash, or of borax and glycerine, may be used. The constitutional symptoms should receive careful attention.

(3) **Ulcerative Stomatitis.**—This form, which is also known by the names of *fetid stomatitis*, or *putrid sore mouth*, occurs particularly in children after the first dentition. It may prevail as a wide-spread epidemic in institutions in which the sanitary conditions are defective. It has been met with in jails and camps. Insufficient and unwholesome food, improper ventilation, and prolonged damp, cold weather seem to be special predisposing causes. Lack of cleanliness of the mouth, the presence of carious teeth, and the collection of tartar around them favor the development of the disease. The affection spreads like a specific disease, but the microbe has not yet been isolated. It has been held that the disease is the same as the foot-and-mouth disease of cattle, and that it is conveyed by the milk, but there is no positive evidence on these points. Payne suggests that the virus is identical with that of contagious impetigo.

The morbid process begins at the margin of the gums, which become swollen and red, and bleed readily. Ulcers form, the bases of which are covered with a grayish-white, firmly adherent membrane. In severe cases the teeth may become loosened and necrosis of the alveolar process may occur. The ulcers extend along the gum-line of the upper and lower jaws; the tongue, lips, and mucosa of the cheeks are usually swollen, but rarely ulcerated. There is salivation, the breath is foul, and mastication is painful. The submaxillary lymph glands are enlarged. The constitutional symptoms are often severe, and in institutions death sometimes results in the case of debilitated children.

In the treatment of this form of stomatitis chlorate of potash has been found to be almost specific. It should be given in doses of ten grains, three times a day, to a child, and to an adult double that amount. Locally it may be used as a mouth-wash, or the powdered salt may be applied directly to the ulcerated surfaces. When there is much fetor a

permanganate-of-potash wash may be used, and an application of nitrate of silver may be made to the ulcers.

There are several *other varieties* of ulcerative sore mouth, which differ entirely from this form. Ulcers of the mouth are common in nursing women, and are usually seen on the mucous membrane of the lips and cheeks. They develop from the mucous follicles, and are from three to five millimetres in diameter. They may cause little or no inconvenience; but in some instances they are very painful and interfere seriously with the taking of food and its mastication. As a rule they heal readily after the application of nitrate of silver, and the condition is an indication for tonics, fresh air, and a better diet.

Parrot describes the occasional appearance in the new-born of small ulcers symmetrically placed on the hard palate on either side of the middle line. They are met with in very debilitated children. The ulcers rarely heal; usually they tend to increase in size, and may involve the bone.

(4) **Parasitic Stomatitis** (*Thrush; Soor; Muguet*).—This affection, most commonly seen in children, is dependent upon a fungus, the *saccharomyces albicans*, called by Robin the *oïdium albicans*. It belongs to the order of yeast fungi, and consists of branching filaments, from the ends of which ovoid torula cells develop. The disease does not arise apparently in a normal mucosa. The use of an improper diet, uncleanliness of the mouth, the acid fermentation of remnants of food, or the development, from any cause, of catarrhal stomatitis predispose to the growth of the fungus. In institutions it is frequently transmitted by unclean feeding-bottles, spoons, etc. It is not confined to children, but is met with in adults in the final stages of fever, in chronic tuberculosis, diabetes, and in cachectic states. The parasite develops in the upper layers of the mucosa, and the filaments form a dense felt-work among the epithelial cells. The disease begins on the tongue and is seen in the form of slightly raised, pearly-white spots, which increase in size and gradually coalesce. The membrane thus formed can be readily scraped off, leaving an intact mucosa, or, if the process extends deeply, a bleeding, slightly ulcerated surface. The disease spreads to the cheeks, lips, and hard palate, and may involve the tonsils and pharynx. In very severe cases the entire buccal mucosa is covered by the grayish-white membrane. It may even extend into the œsophagus and, according to Parrot, to the stomach and cæcum. It is occasionally met with on the vocal cords. Robust, well-nourished children are sometimes affected, but it is usually met with in enfeebled, emaciated infants with digestive or intestinal troubles. In such cases the disease may persist for months.

The affection is readily recognized, and must not be confounded with aphthous stomatitis, in which the ulcers, preceded by the formation of vesicles, are perfectly distinctive. In thrush the microscopical examina-

tion shows the presence of the characteristic fungus throughout the membrane. In this condition, too, the mouth is usually dry—a striking contrast to the salivation accompanying aphthæ.

Thrush is more readily prevented than removed. The child's mouth should be kept scrupulously clean, and, if artificially fed, the bottles should be thoroughly sterilized. Lime-water or any other alkaline fluid, such as the bicarbonate of soda (a drachm to a tumbler of water), may be employed. When the patches are present these alkaline mouth-washes may be continued after each feeding. A spray of borax or of sulphite of soda (a drachm to the ounce) or the black wash with glycerine may be employed. The permanganate of potassium is also useful. The constitutional treatment is of equal importance, and it will often be found that the thrush persists, in spite of all local measures, until the general health of the infant is improved by change of air or the relief of the diarrhœa, or, in obstinate cases, the substitution of a natural for the artificial diet.

(5) **Gangrenous Stomatitis** (*Cancrum Oris; Noma*).—An affection characterized by a rapidly progressing gangrene, starting on the gums or cheeks, and leading to extensive sloughing and destruction. This terrible but fortunately rare disease is seen only in children under very insanitary conditions or during convalescence from the acute fevers. It is more common in girls than in boys. It is met with between the ages of two and five years. In at least one half of the cases the disease has developed during convalescence from measles. Cases have been seen also after scarlet fever and typhoid. The mucous membrane is first affected, usually of the gums or of one cheek. It begins insidiously, and when first seen there is a sloughing ulcer of the mucous membrane, which spreads rapidly and leads to brawny induration of the skin and adjacent parts. The sloughing extends, and in severe cases the cheek is perforated. The disease may spread to the tongue and chin; it may invade the bones of the jaws and even involve the eyelids and ears. In mild cases an ulcer forms on the inner surface of the cheek, which heals or may perforate and leave a fistulous opening. Naturally in such a severe affection the constitutional disturbance is very great, the pulse is rapid, the prostration extreme, and death usually takes place within a week or ten days. The temperature may reach 103° or 104°. Diarrhœa is usually present, and aspiration pneumonia often develops. H. R. Wharton has described a case in which there was extensive colitis. Lingard has found in cases of noma a thread-like bacillus, but its precise relation to the disease is doubtful. The highly refractile bodies described by Sansom in the blood were probably blood-plates.

The treatment of the disease is unsatisfactory. In many cases the onset is so insidious that there is an extensive sloughing sore when the case first comes under observation. Destruction of the sore by the cautery, either the Paquelin or fuming nitric acid, is the most effectual. Antisep-

tic applications should be made to destroy the fetor. The child should be carefully nourished and stimulants given freely.

(6) **Mercurial Stomatitis** (*Ptyalism*).—An inflammation of the mouth and salivary glands caused by mercury, which occurs chiefly in persons who have a special susceptibility, and rarely now as a result of the excessive use of the drug. It is met with also in persons whose occupation necessitates the constant handling of mercury. It often follows the administration of repeated small doses. Thus, a patient with heart disease who was ordered an eighth of a grain of calomel every three hours for diuretic purposes had, after taking eight or ten doses, a severe stomatitis, which persisted for several weeks. I have known it to follow also the administration of small doses of gray powder. The patient complains first of a metallic taste in the mouth, the gums become swollen, red, and sore, mastication is difficult, and soon there is a great increase in the secretion of the saliva, which flows freely from the mouth. The tongue is swollen, the breath has a foul odor, and, if the affection progresses, there may be ulceration of the mucosa, and, in rare instances, necrosis of the jaw. Although troublesome and distressing, the disease is rarely serious, and recovery usually takes place in a couple of weeks. Instances in which the teeth become loosened or detached or in which the inflammation extends to the pharynx and Eustachian tubes are rarely seen now.

The administration of mercury should be suspended so soon as the gums are "touched." Mild cases of the affection subside within a few days and require only a simple mouth-wash. In severer cases the chlorate of potash may be given internally and used to rinse the mouth. The bowels should be freely opened; the patient should take a hot bath every evening and should drink plentifully of alkaline mineral waters. Atropine is sometimes serviceable, and may be given in doses of one one hundredth of a grain twice a day. Iodine is also recommended. When the salivation is severe and protracted the patient becomes much debilitated, anæmia develops, and a supporting treatment is indicated. The diet is necessarily liquid, for the patient finds the chief difficulty in taking food. If the pain is severe a Dover powder may be given at night.

Here may be appropriately mentioned the influence of stomatitis, particularly the mercurial form, upon the developing teeth of children. The condition known as *erosion*, in which the teeth are honeycombed or pitted owing to defective formation of enamel, is indicative as a rule of infantile stomatitis. Such teeth must be distinguished carefully from those of congenital syphilis, which may of course coexist, but the two conditions are distinct. The honeycombing is frequently seen on the incisors; but, according to Jonathan Hutchinson, the test teeth of infantile stomatitis are the first permanent molars, then the incisors, "which are almost as constantly pitted, eroded, and of bad color, often showing the transverse furrow which crosses all the teeth at the same level." Magitot regards these transverse furrows as the result of infantile convulsions or

of severe illnesses during early life. He thinks they are analogous to the furrows on the nails which so often follow a serious disease.

II. DISEASES OF THE SALIVARY GLANDS.

1. **Hypersecretion** (*Ptyalism*).—The normal amount of saliva varies from two to three pints in the twenty-four hours. The secretion is increased during the taking of food and in the physiological processes of dentition. A great increase, to which the term *ptyalism* is applied, is met with under many circumstances. It occurs occasionally in mental and nervous affections and in rabies. Occasionally it is seen in the acute fevers, particularly in small-pox. It has been met with during gestation, usually early, though it may persist throughout the entire course. It has been known to occur at each menstrual period; and, lastly, it is a common effect of certain drugs. Mercury, gold, copper, the iodine compounds, and (among vegetable remedies) jaborandi, muscarin, and tobacco excite the salivary secretion. Of these we most frequently see the effect of mercury in producing ptyalism. The salivation may be present without any inflammation of the mouth.

2. **Xerostomia** (*Arrest of the Salivary and Buccal Secretions; Dry Mouth*).—In this condition, first described by Jonathan Hutchinson, the secretions of the mouth and salivary glands are suppressed. The tongue is red, sometimes cracked, and quite dry; the mucous membrane of the cheeks and of the palate is smooth, shining, and dry; and mastication, deglutition, and articulation are very difficult. The condition is not common. A majority of the cases are in women, and in several instances have been associated with nervous phenomena. The general health, as a rule, is unimpaired. Hadden suggests that it is due to involvement of some centre which controls the secretion of the salivary and buccal glands. A well-marked case came under my observation in a man aged thirty-two, who was sent to me by Donald Baynes on account of a peculiar growth along the gums. This proved to be the remnants of food which, owing to the absence of any salivary or buccal secretions, collected along the gums, became hardened, and adhered to them. The condition lasted for three weeks, and was cured by the galvanic current.*

3. **Inflammation of the Salivary Glands.**

(*a*) *Specific Parotitis.* (See MUMPS.)

(*b*) *Symptomatic parotitis* or *parotid bubo* occurs:

(1) In the course of the infectious fevers—typhus, typhoid, pneumonia, pyæmia, etc. In ordinary practice it occurs oftenest, perhaps, in typhoid fever. It is the result either of septic infection through the blood, or the in-

* Canada Medical and Surgical Journal, vol. v, p. 439, 1877.

flammation, in many cases, passes up the salivary duct and so reaches the gland. The process is usually very intense and leads rapidly to suppuration. It is, as a rule, an unfavorable indication in the course of a fever.

(2) In connection with injury or disease of the abdomen or pelvis, a condition to which Stephen Paget has called special attention. Of 101 cases of this kind, "10 followed injury or disease of the urinary tract, 18 were due to injury or disease of the alimentary canal, and 23 were due to injury or disease of the abdominal wall, the peritonæum, or the pelvic cellular tissue. The remaining 50 were due to injury, disease, or temporary derangement of the genital organs." By temporary derangement is meant slight injuries or natural processes—a slight blow on the testis, the introduction of a pessary, menstruation, or pregnancy. He states that this form of parotitis is not, as a rule, associated with signs of septicæmia or pyæmia. It may occur in connection with gastric ulcer. Of the 101 cases 37 died, the majority of them not from the parotitis, but from the primary lesion with which it was associated. After an operation it occurs usually within the first week, often on the seventh day. There may be pyrexia, but many cases are afebrile. One gland is usually attacked, but both may be involved. In 78 cases in which the termination was noted 45 suppurated and 33 resolved without suppuration. The etiology of this form of parotitis is obscure. Many of the cases are undoubtedly septic.

(3) In association with facial paralysis, as in a case of fatal peripheral neuritis described by Gowers.

In the treatment of parotid bubo the application of half a dozen leeches will sometimes reduce the inflammation and promote resolution. When suppuration seems inevitable hot fomentations should be applied. A free incision should be made *early*.

III. DISEASES OF THE PHARYNX.

(1) **Circulatory Disturbances.**—(*a*) *Hyperæmia* is a common condition in acute and chronic affections of the throat, and is frequently seen as a result of the irritation of tobacco smoke. Venous stasis is seen in valvular disease of the heart, and in mechanical obstruction of the superior vena cava by tumor or aneurism. In aortic insufficiency the capillary pulse may sometimes be seen and the intense throbbing of the internal carotid may be mistaken for aneurism.

(*b*) *Hæmorrhage* is found in association with bleeding from other mucous surfaces, or it is due to local causes in the pharynx itself. In the latter case it may be mistaken for hæmorrhage from the lungs or stomach. The bleeding may come from granulations or vegetations in the naso-pharynx. Sometimes the patient finds the pillow stained in the morning with bloody secretion. The condition is rarely serious, and only

requires suitable local treatment of the pharynx. Occasionally a hæmorrhage takes place into the mucosa, producing a pharyngeal hæmatoma. I have thrice seen a condition of the uvula resembling hæmorrhagic infarction. One was in a patient with acute rheumatism, to whom large doses of salicylic acid had been given; the other two were instances of peliosis rheumatica, in both of which partial sloughing of the uvula took place.

(*c*) *Œdema.*—An infiltrated œdematous condition of the uvula and adjacent parts is not very uncommon in conditions of debility, in profound anæmia, and in Bright's disease. The uvula is sometimes from this cause enormously enlarged, and may lead to difficulty in swallowing or in breathing.

(2) **Acute Pharyngitis** (*Sore Throat; Angina Simplex*).—The entire pharyngeal structures, often with the tonsils, are involved. The condition may follow cold or exposure. In other instances it is associated with constitutional states, such as rheumatism or gout, or with digestive disorders. The patient complains of uneasiness and soreness in swallowing, of a feeling of tickling and dryness in the throat, together with a constant desire to hawk and cough. Frequently the inflammation extends into the larynx and produces hoarseness. Not uncommonly it is only part of a general naso-pharyngeal catarrh. The process may pass into the Eustachian tubes and cause slight deafness. There is stiffness of the neck, the lymph glands of which may be enlarged and painful. The constitutional symptoms are rarely severe. The disease sets in with a chilly feeling and slight fever, and the pulse is increased in frequency. Occasionally the febrile symptoms are more severe, particularly if the tonsils are specially involved. The examination of the throat shows general congestion of the mucous membrane, which is dry and glistening, and in places covered with sticky secretion. The uvula may be much swollen.

Acute pharyngitis lasts only a few days and requires mild measures. If the tonsils are involved and the fever is high, aconite or sodium salicylate may be given. Guaiacum also is beneficial; but in a majority of the cases a calomel purge or a saline aperient and inhalations with steam meet the indications.

(3) **Chronic Pharyngitis.**—This may follow repeated acute attacks. It is very common in persons who smoke or drink to excess, and in those who use the voice very much, such as clergymen, hucksters, and others. It is frequently met with in chronic nasal catarrh. The naso-pharynx and the posterior wall are the parts most frequently affected. The mucous membrane is relaxed, the venules are dilated, and roundish bodies, from two to four millimetres in diameter, reddish in color, project to a variable distance beyond the mucous membrane. These represent the proliferations of lymph tissue about the mucous glands. They may be very abundant, forming elongated rows in the lateral walls of the pharynx. With this there may be a dry glistening state of the

pharyngeal mucosa, sometimes known as *pharyngitis sicca*. The pillars of the fauces, and the uvula are often much relaxed. The secretion forms at the back of the pharynx and the patient may feel it drop down from the vault, or it is tenacious and adherent, and is only removed by repeated efforts at hawking.

In the *treatment*, special attention must be paid to the general health. If possible, the cause should be ascertained. The condition is almost constant in smokers, and cannot be cured without stopping the use of tobacco. The use of food either too hot or too much spiced should be forbidden. When it depends upon excessive exercise of the voice, rest should be enjoined. In many of these cases change of air and tonics help very much. In the local treatment of the throat gargles, washes, and pastilles of various sorts give temporary relief, but when the hypertrophic condition is marked the spots should be thoroughly destroyed by the galvanocautery. In many instances this affords great and permanent relief, but in others the condition persists, and as it is not unbearable, the patient gives up all hope of permanent relief.

(4) **Ulceration of the Pharynx.**—(*a*) Follicular. The ulcers are usually small, superficial, and generally associated with chronic catarrh.

(*b*) Syphilitic ulcers are usually painless, and most frequently situated on the posterior wall of the pharynx. They occur in the secondary stage as small, shallow excavations with the mucous patches. In the tertiary stage the ulcers are due to erosion of gummata, and in healing they leave whitish cicatrices.

(*c*) Tuberculous ulceration is not very uncommon in advanced cases of phthisis, and, if extensive, is one of the most distressing features of the later stages of the disease. The ulcers are irregular, with ill-defined edges and grayish-yellow bases. The posterior wall of the pharynx may have an eroded, worm-eaten appearance. These ulcers are, as a rule, intensely painful.

(*d*) Ulcers occur in connection with pseudo-membranous inflammation, particularly the diphtheritic. In cancer and in lupus ulcers are also present.

(*e*) Ulcers are met with in certain of the fevers, particularly in typhoid.

In many instances the diagnosis of the nature of pharyngeal ulcers is very difficult. The tuberculous and cancerous varieties are readily recognized, but it happens not infrequently that a doubt arises as to the syphilitic character of an ulcer. In many instances the local conditions may be uncertain. Then other evidences of syphilis should be sought for, and the patient should be placed on mercury and iodide of potassium, under which remedies syphilitic ulcers usually heal with great rapidity.

(5) **Acute Infectious Phlegmon of the Pharynx.**—Under this term Senator has described cases in which, along with difficulty in swallowing, soreness of the throat, and sometimes hoarseness, the neck enlarges, the

pharyngeal mucosa becomes swollen and injected, the fever is high, the constitutional symptoms are severe, and the inflammation passes on rapidly to suppuration. The symptoms are very intense. The swelling of the pharyngeal tissues early reaches such a grade as to impede respiration. Very similar symptoms may be produced by the lodgment of foreign bodies in the pharynx.

(6) **Retro-pharyngeal Abscess.**—This may occur as a sequel to one of the fevers, but more commonly results from caries of the cervical vertebræ. It is accompanied with pain in swallowing, sometimes with cough, dyspnœa, and alterations in the character of the voice.

The diagnosis is readily made, as the projecting tumor can be seen, and felt with the finger on the posterior wall of the pharynx.

(7) **Angina Ludovici** (*Ludwig's Angina; Cellulitis of the Neck*).—In medical practice this is seen as a secondary inflammation in the specific fevers, particularly diphtheria and scarlet fever. It may, however, occur idiopathically or result from trauma. It is probably always a streptococcus infection which spreads rapidly from the glands. The swelling at first is most marked in the submaxillary region of one side. The symptoms are, as a rule, intense, and, unless early and thorough surgical measures are employed, there is great risk of systemic infection.

IV. DISEASES OF THE TONSILS.

Apart from the affection of these glands already described in connection with diphtheria, scarlet fever, and syphilis, an acute and a chronic tonsillitis may be recognized.

ACUTE TONSILLITIS.

(1) **Follicular or Lacunar Tonsillitis.**—For practical purposes, under this name may be described the various forms which have been called catarrhal, erythematous, ulcero-membranous, and herpetic.

Etiology.—The disease is met with most frequently in young persons, but in children under ten it is less common than the chronic form. It is rare in infants. Sex has no special influence. Exposure to wet and cold, and bad hygienic surroundings appear to have a direct etiological connection with the disease. In so many instances defective drainage has been found associated with outbreaks of follicular tonsillitis that sewer-gas is regarded as a common exciting cause. One attack renders a patient more liable to subsequent infection. Special stress is laid by some writers upon the coexistence of tonsillitis with rheumatism. Cheadle describes it as one of the phases of rheumatism in childhood with which articular attacks may alternate. I cannot say that, in my experience, the connection

between the two affections has been very striking, except in one point, viz., that an attack of acute rheumatism is not infrequently preceded by inflammation of the tonsils. The existence of pains in the limbs is no evidence of the connection of the affection with rheumatism. A disease so common and wide-spread as acute tonsillitis necessarily attacks many persons in whose families rheumatism prevails or who may themselves have had acute attacks.

Mackenzie gives a table showing that in four successive years more cases occurred in September than in any other month; in October nearly as many; with July, August, and November next. In this country it seems more prevalent in the spring. So many cases develop within a short time that the disease may be almost epidemic. It spreads through a family in such a way that it must be regarded as contagious.

An old notion prevails that there is a definite relation between the tonsils and the testes and ovaries. F. J. Shepherd has called attention to the circumstance that acute tonsillitis is a very common affection in newly married persons. That view is probably correct which regards tonsillitis as a local disease with severe constitutional manifestations, although the fever is often high in proportion to the local symptoms. The commonest organism found in tonsillitis is a streptococcus. Staphylococci also occur. In some cases organisms closely resembling the *bacillus diphtheriæ* of Loeffler have been found, but they do not seem to possess the same malignancy.

Morbid Anatomy.—The lacunæ of the tonsils become filled with exudation products, which form cheesy-looking masses, projecting from the orifices of the crypts. Not infrequently the exudations of contiguous lacunæ coalesce. The intervening mucosa is usually swollen, deep-red in color, and may present herpetic vesicles or, in some instances, even membranous exudation, in which case it may be difficult to distinguish the condition from diphtheria. The creamy contents of the crypt are made up of micrococci and epithelial *débris*.

Symptoms.—Chilly feelings, or even a definite chill, and aching pains in the back and limbs may precede the onset. The fever rises rapidly, and in the case of a young child may reach 105° on the evening of the first day. The patient complains of soreness of the throat and difficulty in swallowing. On examination, the tonsils are seen to be swollen and the crypts present the characteristic creamy exudate. The tongue is furred, the breath is heavy and foul, and the urine is highly colored and loaded with urates. In children the respirations are usually very hurried, and the pulse is greatly increased in rapidity. Swallowing is painful, and the voice often becomes nasal. Slight swelling of the cervical glands is present. In severe cases the symptoms increase and the tonsils become still more swollen. The inflammation gradually subsides, and, as a rule, within a week the fever departs and the local symptoms greatly improve. The tonsils, however, remain somewhat swollen. The prostration and

constitutional disturbance are often out of proportion to the intensity of the local disease.

There are complications which occasionally excite uneasiness. Febrile albuminuria is not uncommon, as Haig-Brown has pointed out. Cases of endocarditis or pericarditis have been found. It is to be borne in mind that in children an apex systolic murmur is by no means uncommon at the height of any fever. The disease may extend to the middle ear. The development of paralytic symptoms, local or general, after an attack which has been regarded as follicular tonsillitis indicates an error in diagnosis.

Diagnosis.—It may be difficult to distinguish follicular tonsillitis from diphtheria. It would seem, indeed, as if there were intermediate forms between the mildest lacunar and the severer pseudo-membranous tonsillitis. In the follicular form the individual yellowish-gray masses, separated by the reddish tonsillar tissue, are very characteristic; whereas in diphtheria the membrane is of ashy gray, and uniform, not patchy. A point of the greatest importance in diphtheria is that the membrane is not limited to the tonsils, but creeps up the pillars of the fauces or appears on the uvula. The diphtheritic membrane when removed leaves a bleeding, eroded surface; whereas the exudation of lacunar tonsillitis is easily separated, and there is no erosion beneath it. In all doubtful cases cultures should, if possible, be made to determine the presence of Loeffler's bacillus.

(2) **Suppurative Tonsillitis.**

Etiology.—This arises under conditions very similar to those mentioned in the lacunar form. It may follow exposure to cold or wet, and is particularly liable to recur. It is most common in adolescence. The inflammation is here more deeply seated. It involves the stroma, and tends to go on to suppuration.

Symptoms.—The constitutional disturbance is very great. The temperature rises to 104° or 105°, and the pulse ranges from 110 to 130. Nocturnal delirium is not uncommon. The prostration may be extreme. There is no local disease of similar extent which so rapidly exhausts the strength of a patient. Soreness and dryness of the throat, with pain in swallowing, are the symptoms of which the patient first complains. One or both tonsils may be involved. They are enlarged, firm to the touch, dusky red and œdematous, and the contiguous parts are also much swollen. The swelling of the glands may be so great that they meet in the middle line, or one tonsil may even push the uvula aside and almost touch the other gland. The salivary and buccal secretions are increased. The glands of the neck enlarge, the lower jaw is fixed, and the patient is unable to open his mouth. In from two to four days the enlarged gland becomes softer, and fluctuation can be distinctly felt by placing one finger on the tonsil and the other at the angle of the jaw. The abscess usually points toward the mouth, but it may point toward the pharynx. It may burst spontaneously, affording instant and great relief. Suffocation has

followed the rupture of a large abscess and the entrance of the pus into the larynx. When the suppuration is peritonsillar and extensive, the internal carotid artery may be opened; but these are, fortunately, very rare accidents.

Treatment.—In the follicular form aconite may be given in full doses. It acts very beneficially in children. The salicylates, given freely at the outset, are regarded by some as specific, but I have seen no evidence of such prompt and decisive action. At night, a full dose of Dover's powder may be given. The use of guaiacum, in the form of two-grain lozenges, is warmly recommended. Iron and quinine should be reserved until the fever has subsided. A pad of spongio-piline or thick flannel dipped in ice-cold water may be applied around the neck and covered with oiled silk. More convenient still is a small ice-bag. Locally the tonsils may be treated with the dry sodium bicarbonate. The moistened finger-tip is dipped into the soda, which is then rubbed gently on the gland and repeated every hour. Astringent preparations, such as iron and glycerine, alum, zinc, and nitrate of silver, may be tried. To cleanse and disinfect the throat, solutions of borax or thymol in glycerine and water may be used.

In suppurative tonsillitis hot applications in the form of poultices and fomentations are more comfortable and better than the ice-bag. The gland should be felt—it cannot always be seen—from time to time, and should be opened when fluctuation is distinct. The progress of the disease may be shortened and the patient spared several days of great suffering if the gland is scarified early. The curved bistoury, guarded nearly to the point with plaster or cotton, is the most satisfactory instrument. The incision should be made from above downward, parallel with the anterior pillar. There are cases in which, before suppuration takes place, the parenchymatous swelling is so great that the patient is threatened with suffocation. In such instances the tonsil must either be excised or tracheotomy or, possibly, intubation performed. Delavan refers to two cases in which he states that tracheotomy would, under these circumstances, have saved life. Patients with this affection require a nourishing liquid diet, and during convalescence iron in full doses.

CHRONIC TONSILLITIS.

(*Chronic Naso-pharyngeal Obstruction; Mouth-Breathing; Aprosexia.*)

Under this heading will be considered also hypertrophy of the adenoid tissue in the vault of the pharynx, sometimes known as the pharyngeal tonsil, as the affection usually involves both the tonsils proper and this tissue, and the symptoms are not to be differentiated.

Chronic enlargement of the tonsillar tissues is an affection of great importance, and may influence in an extraordinary way the mental and bodily development of children.

Etiology.—Hypertrophy of the tonsillar structures is occasionally congenital. Cases are perhaps most frequent in children, during the third hemi-decade. The condition also occurs in young adults, more rarely in the middle-aged. The enlargement may follow diphtheria or the eruptive fevers. The frequency of the occurrence of adenoid growths in the naso-pharynx has been variously stated. Meyer, to whom the profession is indebted for calling attention to the subject, found them in about one per cent of the children in Copenhagen, while Chappell found sixty cases in the examination of two thousand children in New York. These figures give a very moderate estimate of the prevalence of the trouble. It occurs equally in boys and girls, according to some writers with greater prevalence in the former.

Morbid Anatomy.—The tonsils proper present a condition of chronic hypertrophy, due to multiplication of all the constituents of the glands. The lymphoid elements may be chiefly involved without much development of the stroma. In other instances the fibrous matrix is increased, and the organ is then harder, smaller, firmer, and is cut with much greater difficulty.

The adenoid growths, which spring from the vault of the pharynx, form masses varying in size from a small pea to an almond. They may be sessile, with broad bases, or pedunculated. They are reddish in color, of moderate firmness, and contain numerous blood-vessels. "Abundant, as a rule, over the vault, on a line with the fossa of the Eustachian tube, the growths may lie posterior to the fossa—namely, in the depression known as the fossa of Rosenmüller, or upon the parts which are parallel to the posterior wall of the pharynx. The growths appear to spring in the main from the mucous membrane covering the localities where the connective tissue fills in the inequalities of the base of the skull" (Harrison Allen). The growths are most frequently papillomatous with a lymphoid parenchyma. Hypertrophy of the pharyngeal adenoid tissue may be present without great enlargement of the tonsils proper. Chronic catarrh of the nose usually coexists.

Symptoms.—The direct effect of chronic tonsillar hypertrophy is the establishment of mouth-breathing. The indirect effects are deformation of the thorax, changes in the facial expression, and sometimes marked alteration in the mental condition. The establishment of mouth-breathing is the symptom which first attracts the attention. It is not so noticeable by day, although the child may present the vacant expression characteristic of this condition. At night the child's sleep is greatly disturbed; the respirations are loud and snorting, and there are sometimes prolonged pauses, followed by deep, noisy inspirations. The child may wake up in a paroxysm of shortness of breath. Some of these nocturnal attacks may be due to reflex spasm of the glottis.

When the mouth-breathing has persisted for a long time definite changes are brought about in the face, mouth, and chest. The facies is

so peculiar and distinctive that the condition may be evident at a glance. The expression is dull, heavy, and apathetic, due in part to the fact that the mouth is habitually left open. In long-standing cases the child is very stupid-looking, responds slowly to questions, and may be sullen and cross. The lips are thick, the nasal orifices small and pinched-in looking, and in the mouth the superior dental arch is narrowed and the roof considerably raised.

The remarkable alterations in the shape of the chest in connection with enlarged tonsils were first studied by Dupuytren and J. Mason Warren. They are liable to be mistaken for those of rickets. It is the commonest cause of chest deformity in this country. "Anteriorly the ribs are prominent, the sternum is angulated forward at the manubrio-gladiolar junction and grooved at the gladiolo-xiphoid junction. A saucer-shaped depression is often found at the lower costal cartilages. The lower angle of the scapula projects. While the ribs are separated far from each other anteriorly they are so closely pressed together posteriorly, especially at the lower part of the chest, as to have the intercostal spaces practically obliterated" (Harrison Allen). The prominent sternum (chicken breast) with the circular depression in the lateral zones corresponding to the attachment of the diaphragm are the most characteristic features. During sleep, in a chronic mouth-breather, with each inspiration the diaphragm may be seen to draw in the lower and lateral thoracic regions.

The voice is altered and acquires a nasal quality. The pronunciation of certain letters is changed, and there is inability to pronounce the nasal consonants *n* and *m*. Bloch, in his monograph,* lays great stress upon the association of mouth-breathing with stuttering.

The hearing is impaired, usually owing to the extension of inflammation along the Eustachian tube and its obstruction with mucus or the narrowing of its orifice by pressure of the adenoid vegetations. In some instances it may be due to retraction of the drums, as the upper pharynx is insufficiently supplied with air. Naturally the senses of taste and smell are much impaired. With these symptoms there may be little or no nasal catarrh or discharge, but the pharyngeal secretion of mucus is always increased. Children, however, do not notice this, as the mucus is usually swallowed, but older persons expectorate it with difficulty.

Among other symptoms may be mentioned headache, which is by no means uncommon, general listlessness, and an indisposition for physical or mental exertion. Habit-spasm of the face has been described in connection with it. I have known several instances in which permanent relief has been afforded by the removal of the adenoid vegetations. Enuresis is occasionally an associated symptom. The influence upon the mental development is striking. Mouth-breathers are usually dull, stupid, and backward. It is impossible for them to fix the attention for long at a

* Die Pathologie und Therapie der Mundathmung. Wiesbaden, 1889.

time, and to this impairment of the mental function Guye, of Amsterdam, has given the name *aprosexia.* Headaches, forgetfulness, inability to study without discomfort, are frequent symptoms of this condition in students. The practitioner must bear in mind that all of these symptoms may be found in connection with adenoid growths in the vault of the pharynx without especial enlargement of the tonsils, and that both in diagnosis and treatment particular attention must be paid to the former.

A symptom specially associated with enlarged tonsils is fetor of the breath. In the tonsillar crypts the inspissated secretion undergoes decomposition and an odor not unlike that of Rochefort or Limburger cheese is produced. The little cheesy masses may sometimes be squeezed from the crypts of the tonsils. Though the odor may not apparently be very strong, yet if the mass be squeezed between the fingers its intensity will at once be appreciated. In some cases of chronic enlargement the cheesy masses may be deep in the tonsillar crypts; and if they remain for a prolonged period lime salts are deposited and a tonsillar calculus in this way produced.

Children with enlarged tonsils are especially prone to take cold and to recurring attacks of follicular disease. They are also more liable to diphtheria, and in them the anginal features in scarlet fever are always more serious.

Diagnosis.—Enlarged tonsils are readily seen on inspection of the pharynx. There may be no great enlargement of the tonsils and nothing apparent at the back of the throat even when the naso-pharynx is completely blocked with adenoid vegetations. In children the rhinoscopic examination is rarely practicable. Digital examination is the most satisfactory. The growths can then be felt either as small, flat bodies or, if extensive, as velvety, grape-like papillomata.

Treatment.—If the tonsils are large and the general state is evidently influenced by them they should be at once removed. Applications of iodine and iron, or pencilling the crypts with nitrate of silver, are of service in the milder grades, but it is waste of time to apply them in very enlarged glands. There is a condition in which the tonsils are not much enlarged, but the crypts are constantly filled with cheesy secretions and cause a very bad odor in the breath. In such instances the removal of the secretion and thorough pencilling of the crypts with chromic acid may be practised. The galvano-cautery is of great service in many cases of enlarged tonsils when there is any objection to the more radical surgical procedure.

The treatment of the adenoid growths in the pharynx is of the greatest importance, and should be thoroughly carried out. Parents should be frankly told that the affection is serious, one which impairs the mental not less than the bodily development of the child. In spite of the thorough ventilation of this subject by specialists, practitioners do not appear to have grasped as yet the full importance of this disease. They

are far too apt to temporize and to postpone unnecessarily radical measures. The child must be etherized, when the growths can be removed either with the finger-nail, which in most instances is sufficient, or with a suitable curette. Considerable hæmorrhage may follow, but it is usually checked quickly. The good effects of the operation are often apparent within a few days, and the child begins to breathe through the nose. In some instances the habit of mouth-breathing persists. As soon as the child goes to sleep the lower jaw drops and the air is drawn into the mouth. In these cases a chin strap can be readily adjusted, which the child may wear at night. In severe cases it may take months of careful training before the child can speak properly.

Throughout the entire treatment attention should be paid to hygiene and diet, and cod-liver oil and the iodide of iron may be administered with benefit.

V. DISEASES OF THE ŒSOPHAGUS.

I. ACUTE ŒSOPHAGITIS.

Etiology.—Acute inflammation occurs (*a*) in the catarrhal processes of the specific fevers; more rarely as an extension from catarrh of the pharynx. (*b*) As a result of intense mechanical or chemical irritation, produced by foreign bodies, by very hot liquids, or by strong corrosives. (*c*) In the form of pseudo-membranous inflammation in diphtheria, and occasionally in pneumonia, typhoid fever, and pyæmia. (*d*) As a pustular inflammation in small-pox, and, according to Laennec, as a result of a prolonged administration of tartar emetic. (*e*) In connection with local disease, particularly cancer either of the tube itself or extension to it from without. And, lastly, acute œsophagitis, occasionally with ulceration, may occur spontaneously in sucklings.

Morbid Anatomy.—It is extremely rare to see redness of the mucosa, except when chemical irritants have been swallowed. More commonly the epithelium is thickened and has desquamated, so that the surface is covered with a fine granular substance. The mucous follicles are swollen and occasionally there may be seen small erosions. In the pseudo-membranous inflammation there is a grayish croupous exudate, usually limited in extent, at the upper portion of the gullet. This must not be confounded with the grayish-white deposit of thrush in children. The pustular disease is very rare in small-pox. In the phlegmonous inflammation the mucous membrane is greatly swollen, and there is purulent infiltration in the submucosa. This may be limited as about a foreign body, or extremely diffuse. It may even extend throughout a large part of the gullet. Gangrene occasionally supervenes. Birch-Hirschfeld describes a

remarkable case in an hysterical woman, who vomited a long membranous tube which proved, on examination, to be the detached epithelial lining of the œsophagus. Practically, in post-mortem work, there is no portion of the alimentary canal which more rarely shows signs of disease.

Symptoms.—Pain in deglutition is always present in severe inflammation of the œsophagus, and in the form which follows the swallowing of strong irritants may prevent the taking of food. A dull pain beneath the sternum is also present. In the milder forms of catarrhal inflammation there are usually no symptoms. The presence of a foreign body is indicated by dysphagia and spasm with the regurgitation of portions of the food. Later, blood and pus may be ejected. It is surprising how extensive the disease may be in the œsophagus without producing much pain or great discomfort, except in swallowing. The intense inflammation which follows the swallowing of corrosives, when not fatal, gradually subsides, and often leads to cicatricial contraction and stricture.

The *treatment* of acute inflammation of the œsophagus is extremely unsatisfactory, particularly in the severer forms. The slight catarrhal cases require no special treatment. When the dysphagia is intense it is best not to give food by the mouth, but to feed entirely by enemata. Fragments of ice may be given, and as the pain and distress subside, demulcent drinks. External applications of cold often give relief.

A *chronic* form of œsophagitis is described, but it results usually from the prolonged action of the causes which produce the acute form.

Associated with chronic heart disease and more frequently with the senile and the cirrhotic liver, the œsophageal veins may be enormously distended and varicose, particularly toward the stomach. In these cases the mucous membrane is in a state of chronic catarrh, and the patient has frequent eructations of mucus. Rupture of these œsophageal veins may cause fatal hæmorrhage. Two cases of the kind have occurred in my experience.

II. SPASM OF THE ŒSOPHAGUS (*Œsophagismus*).

This so-called spasmodic stricture of the gullet is met with in hysterical patients and hypochondriacs, also in chorea, epilepsy, and especially hydrophobia. It is sometimes associated also with the lodgment of foreign bodies. The idiopathic form is found in females of a marked neurotic habit, but may also occur in elderly men. It may be present only during pregnancy. Of three cases which have come under my observation, two were in men, one a hypochondriac over sixty years of age who for many months had taken only liquid food, and with great difficulty, owing to a spasm which accompanied every attempt to swallow. The readiness with which the bougie passed and the subsequent history showed the true nature of the case. The patient complains of inability to

swallow solid food, and in extreme instances even liquids are rejected. The attack may come on abruptly, and be associated with emotional disturbances and with substernal pain. The bougie, when passed, may be arrested temporarily at the seat of the spasm, which gradually yields, or it may slip through without the slightest effort. The condition is rarely serious. Death has however followed.

The *diagnosis* is not difficult, particularly in young persons with marked nervous manifestations. In elderly persons œsophagismus is almost always connected with hypochondriasis, but great care must be taken to exclude cancer.

In some cases a cure is at once effected by the passage of a bougie. The general neurotic condition also requires special attention.

Paralysis of the œsophagus scarcely demands separate consideration. It is a very rare condition, due most often to central disease, particularly bulbar paralysis. It may be peripheral in origin as in diphtheritic paralysis. Occasionally it occurs also in hysteria. The essential symptom is dysphagia.

III. STRICTURE OF THE ŒSOPHAGUS.

This results from: (*a*) Congenital narrowing. (*b*) The cicatricial contraction of healed ulcers, usually due to corrosive poisons, occasionally to syphilis. (*c*) The growth of tumors in the walls, as in the so-called cancerous stricture. Occasionally polypoid tumors projecting from the mucosa produce great narrowing. (*d*) External pressure by aneurism, enlarged lymph glands, enlarged thyroid, other tumors, and sometimes by pericardial effusion.

The cicatricial stricture may occur anywhere in the gullet, and in extreme cases may, indeed, involve the whole tube, but in a majority of instances it is found either high up near the pharynx or low down toward the stomach. The narrowing may be extreme, so that only small quantities of food can trickle through, or the obstruction may be quite slight. There is usually no difficulty in making a diagnosis of the cicatricial stricture, as the history of mechanical injury or the swallowing of a corrosive fluid makes clear the nature of the case. When the stricture is low down the œsophagus is dilated and the walls are usually much hypertrophied. When it is high in the gullet the food is usually rejected at once, whereas if low it may be retained and a considerable quantity collects before it is regurgitated. Any doubt as to its having reached the stomach is removed by the alkalinity of the materials ejected and the absence of the characteristic gastric odor. Auscultation of the œsophagus may be practised and is sometimes of service. The patient takes a mouthful of water and the auscultator listens along the left of the spine. During deglutition at the seat of the stricture, in place of the normal œsophageal *bruit*, there will be

heard a loud splashing, gurgling sound. The passage of the œsophageal bougie will determine more accurately the locality. Conical bougies attached to a flexible whalebone stem are the most satisfactory, but the gum-elastic stomach tube may be used; a large one should be tried first. The patient should be placed on a low chair with the head well thrown back. The index finger of the left hand is passed far into the pharynx, and in some instances this procedure alone may determine the presence of a new growth. The bougie is passed beside the finger until it touches the posterior wall of the pharynx, then along it, more to one side than in the middle line, and so gradually pushed into the gullet. It is to be borne in mind that in passing the cricoid cartilage there is often a slight obstruction. Great gentleness should be used, as it has happened more than once that the bougie has been passed through a cancerous ulcer into the mediastinum or through a diverticulum. I have known this accident to happen twice—once in the case of a distinguished surgeon, who performed œsophagotomy and passed the tube, as he thought, into the stomach. The post-mortem on the next day showed that the tube had entered a diverticulum and through it the left pleura, in which the milk injected through the tube was found. In the other instance the tube passed through a cancerous ulcer into the lung, which was adherent and inflamed. Fortunately these accidents, sometimes unavoidable, are extremely rare. It is well always, as a precautionary measure before passing the bougie, to examine carefully for aneurism, which may produce all the symptoms of organic stricture. In cases in which the stricture is extreme there is always emaciation.

The prognosis in these cases is good so long as the stricture is dilatable. The persistent treatment of cicatricial stricture by gradual dilatation is very beneficial, and patients improve remarkably under this method. When extreme, the treatment by bougie is not possible, and the question of œsophagotomy or gastrotomy must be considered. Rectal alimentation should be employed whenever the patient is unable to take sufficient food by the mouth.

IV. CANCER OF THE ŒSOPHAGUS.

This is usually epithelioma. It is not an uncommon disease, and occurs more frequently in males than in females. The common situation is in the upper third of the tube. At first confined to the mucous membrane, the cancer gradually increases and soon ulcerates. The lumen of the tube is narrowed, but when ulceration is extensive in the later stages the stricture may be less marked. Dilatation of the tube and hypertrophy of the walls usually take place above the cancer. The cancerous ulcer may perforate the trachea or a bronchus, the lung, the mediastinum, the aorta or one of its larger branches, the pericardium, or it may erode the

vertebral column. In my experience perforation of the lung has been the most frequent, producing, as a rule, local gangrene.

Symptoms.—The earliest symptom is dysphagia, which is progressive and may become extreme, so that the patient emaciates rapidly. Regurgitation may take place at once; or, if the cancer is situated near the stomach, it may be deferred for ten or fifteen minutes, or even longer if the tube is much dilated. The rejected materials may be mixed with blood and may contain cancerous fragments. In persons over fifty years of age persistent difficulty in swallowing accompanied by rapid emaciation usually indicates œsophageal cancer. The cervical lymph glands are frequently enlarged and may give early indication of the nature of the trouble. Pain may be persistent or is present only when food is taken. In certain instances the pain is very great. I saw an autopsy on a case of cancer of the œsophagus in which the patient gradually became emaciated, but had no special symptoms to call attention to the disease. These latent cases are, however, very rare.

The *prognosis* is hopeless, and the patients usually become progressively emaciated, and die either of asthenia or sudden perforation of the ulcer.

In the *diagnosis* of the condition it is important, in the first place, to exclude pressure from without, as by aneurism or other tumor. The history enables us to exclude cicatricial stricture and foreign bodies. The sound may be passed and the presence of the stricture determined. As mentioned above, great care should be exercised.

Treatment.—In most cases milk and liquids can be swallowed, but supplementary nourishment should be given by the rectum. It may be advisable in some instances to pass a tube into the stomach and attempt to feed in this way. If the patient is willing to take the risk, œsophagotomy or gastrotomy may be performed in order to prolong life.

V. RUPTURE OF THE ŒSOPHAGUS.

This may occur in a healthy organ as a result of prolonged vomiting. Boerhaave described the first case in Baron Wassennar, who "broke asunder the tube of the œsophagus near the diaphragm, so that, after the most excruciating pain, the elements which he swallowed passed, together with the air, into the cavity of the thorax, and he expired in twenty-four hours." Fitz has reported a case and has analyzed the literature on the subject up to 1877. The accident has usually occurred during vomiting after a full meal or when intoxicated. It is, of course, invariably fatal.

Much more common is the post-mortem digestion of the œsophagus, which was first described by King, of Guy's Hospital. It is not very infrequent. In one instance I found the contents of the stomach in the left pleura. The erosion is in the posterior wall, and may be of considerable extent.

VI. DILATATIONS AND DIVERTICULA.

Stenosis of the gullet is followed by secondary dilatation of the tube above the constriction and great hypertrophy of the walls. Primary dilatation is extremely rare. The tube may attain extraordinary dimensions—30 cm. in circumference in Luschka's case. Regurgitation of food is the most common symptom. There may also be difficulty in breathing from pressure.

Diverticula are of two forms: (*a*) Pressure diverticula, which are most common at the junction of the pharynx and gullet, on the posterior wall. Owing to weakness of the muscles at this spot, local bulging occurs, which is gradually increased by the pressure of food, and finally forms a saccular pouch. (*b*) The traction diverticula situated on the anterior wall near the bifurcation of the trachea, result, as a rule, from the extension of inflammation from the lymph glands with adhesion and subsequent cicatricial contraction, by which the wall of the gullet is drawn out.

VI. DISEASES OF THE STOMACH.

I. METHODS OF CLINICAL EXAMINATION.

The stomach normally occupies the left upper quadrant of the abdomen, one quarter of the organ only lying to the right of the median line; it is bordered above by the diaphragm and liver, below by the intestine and transverse colon; on the left it reaches the spleen, and on the right it touches the gall-bladder; anteriorly it lies against the ribs and the abdominal wall. The longitudinal axis extends from the left above downward and backward to the right.

The cardiac orifice is about opposite the sternal border of the sixth or seventh left costal cartilage. The highest point of the fundus reaches the level of the fifth rib, or even that of the fourth interspace, while the lowest point is 3 or 4 cm. above the navel. The pylorus lies on a level with the tip of the xiphoid cartilage at a point midway between the right sternal and parasternal lines; it is normally covered by the left lobe of the liver. With the stomach moderately filled with air the upper limit of resonance reaches the fifth interspace in the left mammary line, while the lower limit is several cm. above the navel.

The greatest vertical diameter of gastric resonance varies, according to Pacanowski, from 10 to 14 cm. in the male, and is about 10 cm. in the female.

Methods for determining the Position and Size of the Stomach.—(1) Inflation by bicarbonate of soda and tartaric acid. Dissolve a teaspoonful of each separately in as small a quantity of water as possible,

and let the patient drink the one solution immediately after the other.

(2) Inflation by means of a bulb-syringe apparatus which can be attached to a stomach tube already introduced.

(3) As a makeshift the patient may be given 250 to 500 c. c. of water on an empty stomach in divided doses and the lower limit of the stomach determined by percussion after each drink. The normal stomach sinks gradually to a point a little above the navel, while the dilated and atonic stomach falls rapidly to a much lower level.

The first method is the simplest and most practical, and is generally one of the first steps in the physical examination; the tube is not introduced until the test-meal has been given. The method has the objection that the amount of air introduced cannot be so well regulated and that one may not in a given case fill the stomach to the entire capacity, while occasionally a spasmodic contraction of the cardia and pylorus may give the patient for a time some discomfort.

Auscultation of the Deglutatory Murmurs.—On listening at the tip of the xiphoid cartilage as the patient swallows a mouthful of water one hears normally two murmurs. (1) The primary murmur is heard synchronously with the act of deglutition and sounds as if water were injected into a space containing air. (2) The secondary murmur is heard up to twelve seconds later and is a coarser gurgling sound. It is well while listening to place one hand on the trachea, as the first murmur may be absent. In œsophageal and cardiac stenosis the second sound is delayed and altered in character.

The following description of methods is merely a rough summary. For fuller particulars see the works of Ewald, Boas, Leo, Wesener, etc.

Examination of the Contents of the Stomach.—Various forms of test-meals have been proposed. The simplest and most satisfactory is that of Ewald. His test breakfast (*Probefrühstück*) consists of one roll (*Brödchen*)—about thirty grammes of white bread—and one glass of water or a cup of tea without milk or sugar. One hour later the contents are to be expressed.

The contents should not be more than 20 to 40 c. c. The filtrate should be a clear yellow or yellowish-brown fluid. The fluid should contain free hydrochloric acid; it should not contain sufficient lactic acid to be recognized by the ordinary tests. Pepsin and pepsinogen, the curdling ferment and its zymogen, should be present.

Albuminoids should be almost entirely converted into peptones; propeptones, if present at all, should be recognizable only in traces. Starches should be so far converted into achroödextrin, dextrose, or maltose that the reaction for starch or erythrodextrin with Lugol's solution should be no longer present.

Chemical Examination of the Gastric Contents.

(1) *Acidity* may be determined by litmus paper.

(2) *Presence of Free Acid.*—(*a*) Tropæolin O. O. The brownish-yellow color of the alcoholic solution is turned by the addition of a fluid containing free acid to a deep mahogany brown or brown-red or deep red, according to the strength of the acid. This is most commonly used as tropæolin paper—strips of filter paper soaked for some time in an alcoholic solution. The paper must not be kept too long. It is best to make up a new quantity monthly at least.

(*b*) Congo red. Solutions of Congo red of a brick-red color are turned blue by the addition of a fluid containing pure acid. This is best used as Congo paper, which is a very delicate reagent, and, on the whole, the most satisfactory. Many other reagents have been used (methyl violet, fuchsin, malachite green, benzopurpurin), but the two above-mentioned tests are probably as satisfactory as any.

(3) *Presence of Free HCl.*—The best and simplest test is that of Günzburg: Phloroglucin, 2; vanillin, 1; absolute alcohol, 30. To a drop of the gastric contents (better filtered) add a similar quantity of the reagent on a porcelain plate. On evaporation gradually to dryness over a flame, a beautiful rose-red color begins to appear at the edges if HCl is present. This is merely a test for a free mineral acid, but HCl is the only one present in the gastric juice.

(4) *Presence of Lactic Acid.*—The best test is that of Uffelmann. Add 1 to 2 drops of tinctura ferri chloridi to 10 to 20 c. c. of a 5-per-cent solution of carbolic acid and dilute with water till it assumes an amethyst-blue color. On the addition of a few drops of a solution containing lactic acid to about 1 c. c. of this solution the color changes to a clear lemon-yellow. The test may be simulated in the presence of phosphates, mineral acids in concentration, grape sugar, alcohol, etc.; hence in cases of doubt it is always prudent to shake 20 c. c. of gastric juice with 10 c. c. of ether three times and then evaporate the ether to dryness over a water bath. To the ether residue, which contains any lactic acid present, add several drops of water. On the addition to this of an equal quantity of the reagent a reliable test for lactic acid may be obtained.

(5) *Butyric acid* gives with Uffelmann's reagent a result very similar to that with lactic acid. The color is, however, more brownish. The odor is sufficient evidence of its presence for practical purposes, which is also true of

(6) *Acetic Acid.*

Quantitative Tests.—(*a*) Test for the total acidity. This test is practically a test for the HCl, where this is present to any extent, as, under these circumstances, other acids are present usually in unappreciable quantities. To 5 to 10 c. c. of filtered gastric contents, a one-tenth normal solution of sodic hydrate is added from a burette till neutralization. This point can be determined by adding a drop of an alcoholic solution of phenolphthalein to the gastric juice. The solution remains colorless in acid or neutral solution, but turns red in alkaline. This test estimates not only the

free HCl, but that in combination. Normally 4 to 6 to 6·5 of the one-tenth solution is required. Each c. c. of this one-tenth solution = ·003646 HCl.

(*b*) *Test for Free HCl.*—If one desires to estimate more accurately the free HCl, the simplest method is Boas's modification of that of Mintz. From 10 c. c. of the gastric contents all organic acids are removed by shaking with 100 c. c. of ether, and then the test performed as above until Congo shows no longer a grayish-blue discoloration.

Quantitative tests for organic acids are complicated and in practice unnecessary.

Tests for Pepsin and Curdling Ferment and their Zymogens.—In the presence of free HCl it is unnecessary to examine for these elements, as they may be safely assumed to be present.

(1) *Test for Pepsin and Pepsinogen.*—(*a*) In presence of HCl the presence of pepsin may be determined by adding to 5 to 10 c. c. of the gastric contents a small piece of egg albumen and observing digestion at 37° to 40° during several hours.

(*b*) In the absence of HCl, pepsinogen alone is found. Add to 10 c. c. of the filtered gastric contents 1 to 2 drops of a 25 per cent HCl solution; add, as before, a small shaving of egg albumen, and see if it is dissolved. The HCl turns the pepsinogen into pepsin.

(2) *Test for the Curdling Ferment and its Zymogen.*—(*a*) Test for the curdling ferment. Neutralize exactly 5 to 10 c. c. of the filtered gastric contents with one tenth normal NaOH solution and mix with an equal quantity of neutral or amphoteric milk. If the ferment is present curdling will occur in from ten to fifteen minutes at 37° to 40°. One may proceed more simply by adding 3 to 5 drops of the filtered gastric juice to 10 c. c. of milk, when curdling will occur as above.

(*b*) Test for the zymogen. To 10 c. c. of filtered gastric juice add CaO_2H_2 till slightly alkaline. This sets the zymogen free, and, on mixing with an equal quantity of milk, congulation will occur as above.

These tests are of much value in the absence of HCl to determine the condition of the mucous membrane. For HCl alone may be absent for a greater or less length of time from various nervous causes, while the absence of pepsin and its curdling ferment at the same time would suggest a serious impairment of the secretory functions.

Tests for the condition of the albuminoids in digestion are complicated, and not necessary in an ordinary clinical examination (directions can be found in the books of Ewald, Boas, Lee, von Jaksch, Wesener, etc.).

Tests for the Condition of the Starch.—If, after an hour of digestion, the addition of a drop of Lugol's solution to the filtered gastric juice is followed by the reaction for starch (blue) or erythrodextrin (purple), we may know that the digestion of starch has been hindered. This is usually due to a hyperacidity.

Tests for the Motive Power of the Stomach.—There are various methods, but practically perfectly good results can be obtained by observation

of the amount of fluid obtained after a test breakfast. More than 40 c. c. is a sure indication of motor insufficiency. Large quantities are always suggestive of dilatation.

Test for the Absorptive Power of the Stomach.—Kali iodidi (pure), 0·2 gramme, is taken in a perfectly clean capsule when the stomach is empty. The sputa, tested every two or three minutes with starch and HNO_3, give the blue reaction inside of fifteen minutes in normal cases. The conclusions to be drawn from this test are, however, of little value.

II. ACUTE GASTRITIS

(Simple Gastritis; Acute Gastric Catarrh; Acute Dyspepsia).

Etiology.—Acute gastric catarrh, one of the most common of complaints, occurs at all ages, and is usually traceable to errors in diet. It may follow the ingestion of more food than the stomach can digest, or it may result from taking unsuitable articles, which either themselves irritate the mucosa or, remaining undigested, decompose, and so excite an acute dyspepsia. A frequent cause is the taking of food which has begun to decompose, particularly in hot weather. In children these fermentative processes are very apt to excite acute catarrh of the bowels as well. Another very common cause is the abuse of alcohol, and the acute gastritis which follows a drinking-bout is one of the most typical forms of the disease. The tendency to acute indigestion varies very much in different individuals, and indeed in families. We recognize this in using the expressions a "delicate stomach" and a "strong stomach." Gouty persons are generally thought to be more disposed to acute dyspepsia than others. Acute catarrh of the stomach occurs at the outset of many of the infectious fevers.

Lebert described a special infectious form of gastric catarrh, occurring in epidemic form, and only to be distinguished from mild typhoid fever by the absence of rose spots and swelling of the spleen. Many practitioners still adhere to the belief that there is a form of *gastric fever*, but the evidence of its existence is by no means satisfactory, and certainly a great majority of all cases in this country are examples of mild typhoid.

Morbid Anatomy.—Beaumont's study of St. Martin's stomach showed that in acute catarrh the mucous membrane is reddened and swollen, less gastric juice is secreted, and mucus covers the surface. Slight hæmorrhages may occur or even small erosions. The submucosa may be somewhat œdematous. Microscopically the changes are chiefly noticeable in the mucous and peptic cells, which are swollen and more granular, and there is an infiltration of the intertubular tissue with leucocytes.

Symptoms.—In mild cases the symptoms are those of slight "indigestion"—uncomfortable feeling in the abdomen, headache, depression,

nausea, eructations, and vomiting, which usually gives relief. The tongue is heavily coated and the saliva is increased. In children, there are intestinal symptoms—diarrhœa and colicky pains. The pulse may be slightly increased, but in some instances is less frequent than normal; there is usually no fever. The duration is rarely more than twenty-four hours. In the severer forms the attack may set in with a chill and febrile reaction, in which the temperature rises to 102° or 103°. The tongue is furred, the breath heavy, and vomiting is frequent. The ejected substances, at first mixed with food, subsequently contain much mucus and bile-stained fluids. There may be constipation, but very often there is diarrhœa. The urine presents the usual febrile characteristics, and there is a heavy deposit of urates. The abdomen may be somewhat distended and slightly tender in the epigastric region. Herpes may appear on the lips. The attack may last from one to three days, and occasionally longer. The examination of the vomitus shows, as a rule, absence of the hydrochloric acid, presence of lactic and fatty acids, and marked increase in the mucus.

Diagnosis.—The ordinary afebrile gastric catarrh is readily recognized. The acute febrile form is so similar to the initial symptoms of many of the infectious diseases that it is impossible for a day or two to make a definite diagnosis, particularly in the cases which have come on, so to speak, spontaneously and independently of an error in diet. Some of these resemble closely an acute infection; the symptoms may be very intense, and if, as sometimes happens, the attack sets in with severe headache and delirium the case may be mistaken for meningitis. When the abdominal pains are intense the attack may be confounded with gall-stone colic. In discriminating between acute febrile gastritis and the abortive forms of typhoid fever it is to be borne in mind that in the former the temperature rises abruptly, the remissions are slighter, and the drop is more sudden. The initial bronchitis, the well-marked splenic enlargement, and the rose spots are not present. It is a very common error to class under gastric fever the mild forms of the various infectious disorders.

Treatment.—Mild cases recover spontaneously in twenty-four hours, and require no treatment other than a dose of castor oil in children or of blue mass in adults. In the severer forms, if there is much distress in the region of the stomach, the vomiting should be promoted by warm water or the simple emetics. A full dose of calomel, eight to ten grains, should be given, and followed the next morning by a dose of Hunyadi-Janos or Carlsbad water. If there is eructation of acid fluid, bicarbonate of soda and bismuth may be given. The stomach should have, if possible, absolute rest, and it is a good plan in the case of strong persons, particularly in those addicted to alcohol, to cut off all food for a day or two. The patient may be allowed soda water and ice freely. It is well not to attempt to check the vomiting unless it is excessive and protracted. Recovery is

usually complete, though repeated attacks may lead to subacute gastritis or to the establishment of chronic dyspepsia.

Phlegmonous Gastritis; Acute Suppurative Gastritis.—This is an excessively rare disease, characterized by the occurrence of suppurative processes in the submucosa. The affection is more common in men than in women. The cause is seldom obvious. It has been met with as an idiopathic affection, but it has occurred also in puerperal fever and other septic processes, and has occasionally followed trauma. Anatomically there appear to be two forms, a diffuse purulent infiltration and a localized abscess formation, in which case the tumor may reach the size of an egg, and may burst into the stomach or into the peritoneal cavity.

The *symptoms* are variable. There are usually pain in the abdomen, fever, dry tongue, and symptoms of a severe infective process, delirium and coma preceding death. Jaundice has been met with in some instances. Occasionally, when the abscess tumor is large, it has been felt externally, in one case forming a mass as large as two fists. There are instances which run a more chronic course, with pains in the abdomen, fever, and chills.

The *diagnosis* is rarely possible, even when with abscess rupture occurs, and the pus is vomited, as it is not possible to differentiate this condition from an abscess perforating into the stomach from without. It is stated, however, that Chvostek made the diagnosis in one of his cases.

Toxic Gastritis.—This most intense form of inflammation of the stomach is excited by the swallowing of concentrated mineral acids or strong alkalies, or by such poisons as phosphorus, corrosive sublimate, ammonia, arsenic, etc. In the non-corrosive poisons, such as phosphorus, arsenic, and antimony, the process consists of an acute degeneration of the glandular elements, and hæmorrhage. In the powerful concentrated poisons the mucous membrane is extensively destroyed, and may be converted into a brownish-black eschar. In the less severe grades there may be areas of necrosis surrounded by inflammatory reaction, while the submucosa is hæmorrhagic and infiltrated. The process is of course more intense at the fundus, but the active peristalsis may drive the poison through the pylorus into the intestine.

The *symptoms* are intense pain in the mouth, throat, and stomach, salivation, great difficulty in swallowing, and constant vomiting, the vomited materials being bloody and sometimes containing portions of the mucous membrane. The abdomen is tender, distended, and painful on pressure. In the most acute cases symptoms of collapse supervene; the pulse is weak, the skin pale and covered with sweat; there is restlessness, and sometimes convulsions. There may be albumen or blood in the urine, and petechiæ may develop on the skin. When the poison is less intense, the sloughs may separate, leaving ulcers, which too often lead, in the œsophagus, to stricture, and in the stomach to chronic atrophy, and finally to death from exhaustion.

The *diagnosis* of toxic gastritis is usually easy, as inspection of the mouth and pharynx shows, in many instances, corrosive effects, while the examination of the vomit may indicate the nature of the poison.

In poisoning by acids, magnesia should be administered in milk or with egg albumen. When strong alkalies have been taken, the dilute acids should be administered. For the severe inflammation which follows the swallowing of the stronger poisons palliative treatment is alone available, and morphia may be freely employed to allay the pain.

Diphtheritic or Membranous Gastritis.—This condition is met with occasionally in diphtheria, but more commonly as a secondary process in typhus or typhoid fever, pneumonia, pyæmia, small-pox, and occasionally in debilitated children. An instance of it came under my notice in pneumonia. The exudation may be extensive and uniform or in patches. The condition is not recognizable during life.

Mycotic and Parasitic Gastritis.—It occasionally happens that fungi develop in the stomach and excite inflammation. One of the most remarkable cases of the kind is that reported by Kundrat, in which the favus fungus developed in the stomach and intestine.

In cancer and in dilatation of the stomach the sarcinæ and yeast fungi probably aid in maintaining the chronic gastritis. As a rule, the gastric juice is capable of killing the ordinary bacteria. Orth states that the anthrax bacilli, in certain cases, produce swelling of the mucosa and ulceration. Klebs has described a *bacillus gastricus* which develops in the tubules and produces numerous spores, and Eug. Fraenkel has reported a case of acute emphysematous gastritis probably of mycotic origin. The larvæ of certain insects may excite gastritis, as in the cases reported by Gerhardt, Meschede, and others. In rare instances tuberculosis and syphilis attack the gastric mucosa.

III. CHRONIC GASTRITIS

(*Chronic Catarrh of the Stomach; Chronic Dyspepsia*).

Definition.—A condition of disturbed digestion associated with increased mucus formation, qualitative or quantitative changes in the gastric juice, enfeeblement of the muscular coats, so that the food is retained for an abnormal time in the stomach; and, finally, with alterations in the structure of the mucosa.

Etiology.—The causes of chronic gastritis may be classified as follows: (1) Dietetic. The use of unsuitable or improperly prepared food. The persistent use of certain articles of diet, such as very fat substances or foods containing too much of the carbohydrates. The use in excess of tea or coffee, and, above all, alcohol in its various forms. Under this heading, too, may be mentioned the habits of eating at irregular hours or too rapidly and imperfectly chewing the food. A common cause of chronic

catarrh is drinking too freely of ice-water during meals, a practice which plays no small part in the prevalence of dyspepsia in America. Another frequent cause is the abuse of tobacco. (2) Constitutional causes. Anæmia, chlorosis, chronic tuberculosis, gout, diabetes, and Bright's disease are often associated with chronic gastric catarrh. (3) Local conditions: (*a*) of the stomach, as in cancer, ulcer and dilatation, which are invariably accompanied by catarrh; (*b*) conditions of the portal circulation, causing chronic engorgement of the mucous membrane, as in cirrhosis, chronic heart disease, and certain chronic lung affections.

Morbid Anatomy.—Anatomically two forms of chronic gastritis may be recognized, the simple and the sclerotic.

(*a*) **Simple Chronic Gastritis.**—The organ is usually enlarged, the mucous membrane pale gray in color, and covered with closely adherent, tenacious mucus. The veins are large, patches of ecchymosis are not infrequently seen, and in the chronic catarrh of portal obstruction and of chronic heart disease small hæmorrhagic erosions. Toward the pylorus the mucosa is not infrequently irregularly pigmented, and presents a rough, wrinkled, mammillated surface, the *état mammeloné* of the French, a condition which may sometimes be so prominent that writers have described it as *gastritis polyposa*. The membrane may be thinner than normal, and much firmer, tearing less readily with the finger-nail. Ewald thus describes the histological changes: The minute anatomy shows the picture of a parenchymatous and an interstitial inflammation. The gland cells are in part eroded or show cloudy granular swelling or atrophy. The distinction between the "haupt" and "beleg" cells cannot be recognized, and in many places, particularly in the pyloric region, the tubes have lost their regular form and show in many places an atypical branching, like the fingers of a glove. Individual glands are cut off toward the fundus, but appear at the border of the submucosa as cysts, partly empty, with a smooth membrane, partly filled with remnants of hyaline and refractile epithelium. An abundant small-celled infiltration presses apart the tubules and is particularly marked toward the surface of the mucosa, and from the submucosa extensions of the connective tissue may be seen passing between the glands. The mucoid transformation of the cells of the tubules is a striking feature in the process and may extend to the very fundus of the glands.

(*b*) **Sclerotic Gastritis.**—As a final result of the parenchymatous and interstitial changes the mucous membrane may undergo complete atrophy, so that but few traces of secreting substance remain. There appear to be two forms of this sclerotic atrophy—one with thinning of the coats of the stomach, *phthisis ventriculi*, and a retention or even increase of the size of the organ; the other with enormous thickening of the coats and great reduction in the volume of the organ, the condition which is usually described as *cirrhosis ventriculi*. Extreme atrophy of the mucous membrane of the stomach has been carefully studied by Fenwick,

Ewald, and others, and we now recognize the fact that there may be such destruction and degeneration of the glandular elements by a progressive development of interstitial tissue that ultimately scarcely a trace of secreting tissue remains. In a characteristic case, studied by Henry and myself,* the greater portion of the lining membrane of the stomach was converted into a perfectly smooth, cuticular structure, showing no trace whatever of glandular elements, with enormous hypertrophy of the muscularis mucosæ, and here and there formation of cysts. In the other form, with identical atrophy and cyst formation, there is enormous increase in the connective tissue, and the stomach may be so contracted that it does not hold more than a couple of ounces. The walls may measure from two to three centimetres; the greatest increase in thickness is in the submucosa, but the hypertrophy also extends to the muscular layers. While one is not justified in saying that all cases of cirrhosis of the stomach represent a final stage in the history of a chronic catarrh, it is true that in most cases the process is associated with atrophy of the gastric mucosa, while the history indicates the existence of chronic dyspepsia.

Symptoms.—The affection persists for an indefinite period, and, as is the case with most chronic diseases, changes from time to time. The appetite is variable, sometimes greatly impaired, at others very good. Among early symptoms are feelings of distress or oppression after eating, which may become aggravated and amount to actual pain. When the stomach is empty there may also be a painful feeling. The pain differs in different cases, and may be trifling or of extreme severity. When localized and felt beneath the sternum or in the præcordial region it is known as heart-burn or sometimes cardialgia. There is pain on pressure over the stomach, usually diffuse and not severe. The tongue is coated, and the patient complains of a bad taste in the mouth. The tip and margin of the tongue are very often red. Associated with this catarrhal stomatitis there may be an increase in the salivary and pharyngeal secretions. Nausea is an early symptom, and is particularly apt to occur in the morning hours. It is not, however, nearly so constant a symptom in chronic gastritis as in cancer of the stomach, and in mild grades of the affection it may not occur at all. Eructation of gas, which may continue for some hours after taking food, is a very prominent feature in cases of so-called flatulent dyspepsia, and there may be marked distension of the intestines. With the gas, bitter fluids may be brought up. In other instances a clear watery fluid is ejected (pyrosis or water-brash). The vomiting does not often occur when the stomach is empty, but either immediately after eating or an hour or two later. The vomitus consists of food in various stages of digestion and slimy mucus, and the chemical examination shows the presence of abnormal acids, such as butyric, or even acetic, in addition to lactic acid, while the hydrochloric acid, if indeed it is present,

* American Journal of the Medical Sciences, 1886.

is much reduced in quantity. The digestion may be much delayed, and on washing out the stomach as late as seven hours after eating, portions of food are still present. The prolonged retention favors decomposition, the stomach becomes distended with gas, and this, with the chronic catarrh, may induce gradually an atony of the muscular walls. The absorption is slow, and iodide of potassium, given in capsules, which should normally reach the saliva within fifteen minutes, may not be evident for more than half an hour.

Constipation is usually present, but in some instances there is diarrhœa, and undigested food passes rapidly through the bowels. The urine is often scanty, high-colored, and deposits a heavy sediment of urates.

Of other symptoms headache is common, and the patient feels constantly out of sorts, indisposed for exertion, and low-spirited. In aggravated cases melancholia may develop. Trousseau called attention to the occurrence of vertigo, a marked feature in certain cases. The pulse is small, sometimes slow, and there may be palpitation of the heart. Fever does not occur. Cough is sometimes present, but the so-called stomach cough of chronic dyspeptics is in all probability dependent upon pharyngeal irritation.

The symptoms of atrophy of the mucous membrane of the stomach, with or without contraction of the organ, are very complex, and cannot be said to present a uniform picture. The majority of the cases present the symptoms of an aggravated chronic dyspepsia, often of such severity that cancer is suspected. In one of the cases which I examined the persistent distress after eating, the vomiting, and the gradual loss of flesh and strength, very naturally led to this diagnosis, but the duration of the disease far exceeded that of ordinary carcinoma. In the cirrhotic form the tumor mass may sometimes be felt. In atrophy of the stomach, whether associated with cirrhosis or not, the clinical picture may be that of pernicious anæmia. As early as 1860, Flint called attention to this connection between atrophy of the gastric tubules and anæmia, an observation which Fenwick and others have amply confirmed.

Diagnosis.—The use of the stomach-tube and the chemical examination of the contents of the stomach obtained in this way have given us special information with reference to the various forms of gastritis and the modes of differentiating them. The soft-rubber stomach-tube, provided with a funnel-shaped dilatation, is the most satisfactory to use, as it is very readily passed, and if used by the patient is not likely to cause damage. It should be open at the end and possess one or two lateral openings.

Ewald distinguishes three forms of chronic gastritis: (1) Simple gastritis; (2) mucous (*schleimige*) gastritis; (3) atrophy.

In (1) the fasting stomach contains only a small quantity of a slimy fluid, while after the test breakfast the HCl is diminished in quantity and lactic acid and the fat acids are usually present.

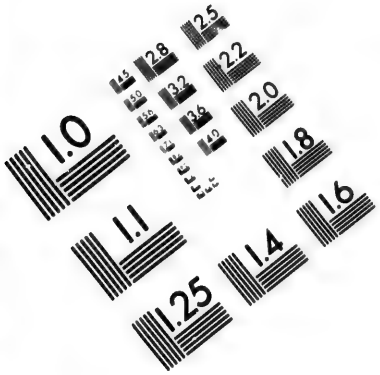
In (2) the acidity is always slight and the condition is distinguished from (1) chiefly by the large amount of mucus present.

In (3) the fasting stomach is generally empty, while after the test breakfast HCl, pepsin, and the curdling ferment are wholly wanting.

Treatment.—When possible the cause in each case should be ascertained and an attempt made to determine the special form of indigestion. Usually there is no difficulty in differentiating the ordinary catarrhal and the nervous varieties. A careful study of the phenomena of digestion in the way already laid down, though not essential in every instance, should certainly be carried out in the more obstinate and obscure forms. Two important questions should be asked of every dyspeptic—first, as to the time taken at his meals; and, second, as to the quantity he eats. Practically a large majority of all cases of disturbed digestion come from hasty and imperfect mastication of the food and from overeating. Especial stress should be laid upon the former point. In some instances it will alone suffice to cure dyspepsia if the patient will count a certain number before swallowing each mouthful. The second point is of even greater importance. People habitually eat too much, and it is probably true that a greater number of maladies arise from excess in eating than from excess in drinking. Particularly is this the case in America, where the average man is abstemious in the matter of alcohol, but imprudent to a degree in all matters relating to food. Moreover, people have not had time to learn the art of cooking, and much of the indigestion, particularly in the country districts, may be charged to the barbarous methods of preparing the food. The treatment may be considered under the headings of dietetic and medicinal.

(*a*) *General and Dietetic.*—A careful and systematically arranged dietary is the first, sometimes the only essential in the treatment of a case of chronic dyspepsia. It is impossible to lay down rules applicable to all cases. Individuals differ extraordinarily in their capability of digesting different articles of food, and there is much truth in the old adage, "One man's food is another man's poison." The individual preferences for different articles of food should be permitted in the milder forms. Physicians have probably been too arbitrary in this direction, and have not yielded sufficiently to the intimations given by the appetite and desires of the patient.

A rigid milk diet may be tried in obstinate cases. Much depends upon whether the patient is able to take and digest milk properly. In the forms associated with Bright's disease and chronic portal congestion, as well as in many instances in which the dyspepsia is part of a neurasthenic or hysterical trouble, this plan in conjunction with rest is most efficacious. If milk is not digested well it may be diluted one third with soda water or Vichy, or five to ten grains of carbonate of soda, or a pinch of salt may be added to each tumblerful. In many cases the milk from which the cream has been taken is better borne. Buttermilk is particularly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suitable, but can rarely be taken for as long a time alone, as patients tire of it much more readily than they do of ordinary milk. Not only can the general nutrition be maintained on this diet, but patients sometimes increase in weight, and the unpleasant gastric symptoms disappear entirely. It should be given at fixed hours and in definite quantities. A patient may take six or eight ounces every three hours. The amount necessary varies a good deal, but at least three to five pints should be given in the twenty-four hours. This form of diet is not, as a rule, well borne when there is a tendency to dilatation of the stomach. The milk may be previously peptonized, but it is impossible to feed a chronic dyspeptic in this way. The stools should be carefully watched, and if more milk is taken than can be digested it is well to supplement the diet with eggs and dry toast or biscuits.

In a large proportion of the cases of chronic indigestion it is not necessary to annoy the patient with such strict dietaries. It may be quite sufficient to cut off certain articles of food. Thus, if there are acid eructations or flatulency, the farinaceous foods should be restricted, particularly potatoes and the coarser vegetables. A fruitful source of indigestion is the hot bread which, in different forms, is regarded as an essential part of an American breakfast. This, as well as the various forms of pancakes, pies and tarts, with heavy pastry, and fried articles of all sorts, should be strictly forbidden. As a rule, white bread, toasted, is more readily digested than bread made from the whole meal. Persons, however, differ very much in this respect, and the Graham or brown bread is for many people most digestible. Sugar and very sweet articles of food should be taken in great moderation or avoided altogether by persons with chronic dyspepsia. Many instances of aggravated indigestion have come to my notice due to the prevalent practice of eating largely of ice-cream. One of the most powerful enemies of the American stomach in the present day is the soda-water fountain, which has usurped so important a place in the apothecary shop.

Fats, with the exception of a moderate amount of good butter, very fat meats, and thick, greasy soups should be avoided. Ripe fruit in moderation is often advantageous, particularly when cooked. Bananas are not, as a rule, well borne. Strawberries are to many persons a cause of an annual attack of indigestion and sore throat in the spring months.

As stated, in the matter of special articles of food it is impossible to lay down rigid rules, and it is the common experience that one patient with indigestion will take with impunity the very articles which cause the greatest distress to another.

Another detail of importance which may be mentioned in this connection is the general hygienic management of dyspeptics. These patients are often introspective, dwelling in a morbid manner on their symptoms, and much inclined to take a despondent view of their condition. Very little progress can be made unless the physician gains

their confidence from the outset. Their fears and whims should not be made too light of or ridiculed. Systematic exercise, carefully regulated, particularly when, as at watering places, it is combined with a restricted diet, is of special service. Change of air and occupation, a prolonged sea voyage, or a summer in the mountains will sometimes cure the most obstinate dyspepsia.

(*b*) *Medicinal.*—The special therapeutic measures may be divided into those which attempt to replace in the digestive juices important elements which are lacking and those which stimulate the weakened action of the organ. In the first group come the hydrochloric acid and ferments, which are so freely employed in dyspepsia. The former is the most important. It is the ingredient in the gastric juice most commonly deficient. It is not only necessary for its own important actions, but its presence is intimately associated with that of the pepsin, as it is only in the presence of a sufficient quantity that the pepsinogen is converted into the active digestive ferment. It is best given as the dilute acid taken in somewhat larger quantities than are usually advised. Ewald recommends large doses—of from 90 to 100 drops—at intervals of fifteen minutes after the meals. Leube and Riegel advise smaller doses. Probably from 15 to 20 drops is sufficient. The prolonged use of it does not appear to be in any way hurtful. The use, however, should be restricted to cases of neurosis and atrophy of the mucous membrane. In actual gastritis its value is doubtful.

The digestive ferments: These are extensively employed to strengthen the weakened gastric and intestinal secretions. The use of pepsin, according to Ewald, may be limited to the cases of advanced mucous catarrh and the instances of atrophy of the stomach, in which it should be given, in doses of from 10 to 15 grains, with dilute hydrochloric acid a quarter of an hour after meals. It may be used in various different forms, either as a powder or in solution or given with the acid. The powder is much more certain. Pepsin wine is generally inert, as there is little of the ferment taken up by alcohol. It is important to use a reliable article. Much that is in the market is valueless.

Pancreatin is of equal or even greater value than the pepsin. Pains should be taken to use a good article, such as that prepared by Merck. It should be given in doses of from 15 to 20 grains, in combination with bicarbonate of soda. It is conveniently administered in tablets, each of which contains 5 grains of the pancreatin and the soda, and of these two or three may be taken fifteen or twenty minutes after each meal. Ptyalin and diastase are particularly indicated when the acid is excessive. The action of the former continues in the stomach during normal digestion. The malt diastase is often very serviceable given with alkalies.

Of measures which stimulate the glandular activity in chronic dyspepsia lavage is by far the most important, particularly in the forms characterized by the secretion of a large quantity of mucus. Luke-warm

water should be used, or, if there is much mucus, a one per cent salt solution, or a three to five per cent solution of bicarbonate of soda. If there is much fermentation the three per cent solution of boric acid may be used, or a dilute solution of carbolic acid. It is best employed in the morning on an empty stomach, or in the evening some hours after the last meal. It is perhaps preferable in the morning, except in those cases in which there is much nocturnal distress and flatulency. Once a day is, as a rule, sufficient, or, in the case of delicate persons, every second day. The irrigation may be continued until the water which comes away is quite clear. It is not necessary to remove all the fluid after the irrigation.

While perhaps in some hands this measure has been carried to extremes, it is one of such extraordinary value in certain cases that it should be more widely employed by practitioners. When there is an insuperable objection to lavage a substitute may be used in the form of warm alkaline drinks, taken slowly in the early morning or the last thing at night.

Of medicines which stimulate the gastric secretion the most important are the bitter tonics, such as quassia, gentian, columbo, cundurango, ipecacuanha, strychnia, and cardamoms. These are probably of more value in chronic gastritis than the hydrochloric acid. Of these strychnia is the most powerful, though none of them have probably any very great stimulating action on the secretion, and influence rather the appetite than the digestion. Of stomachics which are believed to favorably influence digestion the most important are alcohol and common salt. The former would appear to act in moderate quantities by increasing the acid in the gastric juice, and with it probably the pepsin formation. Others hold that it is not so much the secretory as the motor function of the stomach which the alcohol stimulates. In moderate quantities it has certainly no directly injurious influence on the digestive processes. Special care should be taken, however, in ordering alcohol to dyspeptics. If a patient has been in the habit of taking beer or light wines or stimulants with his meals, the practice may be continued if moderate quantities are taken. Beer, as a rule, is not well borne. A dry sherry or a glass of claret is preferable. In the case of women with any form of dyspepsia stimulants should be employed with the greatest caution, and the practitioner should know his patient well before ordering alcohol.

The importance of salt in gastric digestion rests upon the fact that its presence is essential in the formation of the hydrochloric acid. An increase in its use may be advised in all cases of chronic dyspepsia in which the acid is defective.

Treatment of Special Conditions.—Fermentation and flatulency. When the digestion is slow or imperfect, fermentation goes on in the contents, with the formation of gas and the production of lactic, butyric, and acetic acids. For the treatment of this condition careful dieting may suffice, particularly forbidding such articles as tea, pastry, and the coarser vegetables. It is usually combined with pyrosis, in which the

acid fluids are brought into the mouth. Bismuth and carbonate of soda sometimes suffice to relieve the condition. Thymol, creosote, and carbolic acid may be employed. For acid dyspepsia Sir William Roberts recommends the bismuth lozenge of the British Pharmacopœia, the antacid properties of which depend on chalk and bicarbonate of soda. It should be taken an hour or two after meals, and only when the pain and uneasiness are present. Glycerine in from twenty to sixty minim doses, the essential oils, animal charcoal alone or in combination with compound cinnamon powder, may be tried. If there is much pain, chloroform in twenty-minim doses or a teaspoonful of Hoffman's anodyne may be used. If obstinate, lavage is indicated and is sometimes striking in its effects. Alkaline solutions may be used.

Vomiting is not a feature which often calls for treatment in chronic dyspepsia; sometimes in children it is a persistent symptom. Creosote and carbolic acid in drop doses, a few drops of chloroform or of dilute hydrocyanic acid, cocaine, bismuth, and oxalate of cerium may be used. If obstinate, the stomach should be washed out daily.

Constipation is a frequent and troublesome feature of most forms of indigestion. Occasionally small doses of mercury, podophyllin, the laxative mineral waters, sulphur, and cascara may be employed. Glycerine suppositories or the injection of from half a teaspoonful to a teaspoonful of glycerine is very efficacious.

Many cases of chronic dyspepsia are greatly benefited by the use of mineral waters, particularly a residence at the springs with a careful supervision of the diet and systematic exercise. The strict *régime* of certain German Spas is particularly advantageous in the cases in which the chronic dyspepsia has resulted from excess in eating and in drinking. Kissingen, Carlsbad, Ems, and Wiesbaden are to be specially recommended.

IV. NEUROSES OF THE STOMACH.

(1) **Gastralgia; Gastrodynia.**—Severe pains in the epigastrium, paroxysmal in character, occur (*a*) as a manifestation of a functional neurosis, independent of organic disease, and usually associated with other nervous symptoms (it is this form which will here be described); (*b*) in chronic disease of the nervous system, forming the so-called gastric crises; and (*c*) in organic disease of the stomach, such as ulcer or cancer.

The functional neurosis occurs chiefly in women, very commonly in connection with disturbed menstrual function or with pronounced hysterical symptoms. The affection may set in as early as puberty, but it is more common at the menopause. Anæmic, constipated women who have worries and anxieties at home are most prone to the affection. It is more frequent in brunettes than in blondes. Attacks of it sometimes occur in robust, healthy men. More often it is only one feature in a condition of

general neurasthenia or a manifestation of that form of nervous dyspepsia in which the gastric juice or hydrochloric acid is secreted in excess. I am very skeptical as to the existence of a gastralgia of purely malarial origin.

The symptoms are very characteristic; the patient is suddenly seized with agonizing pains in the epigastrium, which pass toward the back and around the lower ribs. The attack is usually independent of the taking of food, and may recur at definite intervals, a periodicity which has given rise to the supposition in some cases that the affection is due to malaria. The most marked periodicity, however, may be in the gastralgic attacks of ulcer. They frequently come on at night. Vomiting is rare; more commonly the taking of food relieves the pain. To this, however, there are striking exceptions. Pressure upon the epigastrium commonly gives relief, but deep pressure may be painful. It seems scarcely necessary to separate the forms, as some have done, into irritative and depressive, as the cases insensibly merge into each other. Stress has been laid upon the occurrence of painful points, but they are so common in neurasthenia that very little importance can be attributed to them.

The *diagnosis* offers many difficulties. Organic disease either of the stomach or of the nervous system must be excluded. In the case of ulcer or cancer this is not always easy. I well remember the case of a poor fellow who was discharged from the Montreal General Hospital as a malingerer. He had been a soldier, was well nourished, had no vomiting, but had severe attacks of abdominal pain. The examination was negative, and it was thought to be a case of simulation. A week subsequent to his discharge he was readmitted with peritonitis from perforation. The fact that the pain is most marked when the stomach is empty and is relieved by the taking of food is sometimes regarded as pathognomonic of simple gastralgia, but to this there are many exceptions, and in cancer the pains may be relieved on eating. The prolonged intervals between the attacks and their independence of diet are important features in simple gastralgia; but in many instances it is less the local than the general symptoms of the case which enable us to make the diagnosis.

(2) **Nervous Dyspepsia.**—According to Leube, who first separated it from the ordinary gastric catarrh, nervous dyspepsia is characterized by sensations of distress and uneasiness during digestion, and yet the act is accomplished within the physiological time limit. The studies of Ewald, Oser, Rosenbach, and others have greatly extended our knowledge of the condition. The cases are met with most frequently in those who have either inherited a neurotic constitution or have gradually, through indiscretions, brought about a condition of nervous prostration. All grades occur, from the emaciated, skeleton-like subject of anorexia nervosa to the well-nourished, healthy-looking, fresh-complexioned patient whose constant complaint is distress and uneasiness after eating. If in a case of dyspepsia the stomach is found empty seven hours after the test dinner, the supposition is that the trouble is nervous (Leube). The separation of

the different forms can only be made accurately by the chemical examination of the juices.

Clinical Forms.—Leube recognizes three chief types. (*a*) Nervous dyspepsia with normal secretion. There is no dilatation of the stomach, no pain on pressure, and no change in the condition of the acid. The test meal is digested within the normal time. Yet, despite the fact that the motor and chemical functions of the organ are perfectly performed, there are distress and uneasiness during the act of digestion. The patient complains of pressure and distention of the stomach; eructations occur.

(*b*) The condition of subacidity or inacidity. Lack of the normal amount of acid is found in chronic catarrh, and particularly in cancer. According to Leube, reduction in the normal amount of acid may exist with the most pronounced symptoms of nervous dyspepsia, and yet the stomach will be free from food within the regular time. A condition in which the gastric juice is entirely without acid may occur in cancer, in extreme sclerosis of the mucous membrane, and as a nervous manifestation of hysteria, and occasionally of tabes. The most aggravated cases are those associated with hysteria and neurasthenia. In addition to the general symptoms, there are loss of appetite, sleeplessness, and gastric distress, and when the stomach is empty there are uneasy local sensations and general feelings of malaise, headache, and dizziness.

(*c*) Nervous dyspepsia with hyperacidity of the gastric juices. This is a form of dyspepsia which has long been recognized, but of late has been specially studied by Reichman and others. The percentage of acid may be doubled. This increase in the acid may be an intermittent condition or continuous. The periodic form is really a neurosis of secretion—*gastroxynsis* of Rosenbach—which may be quite independent of the time of digestion. Such cases are rare and are associated either with profound neurasthenia or with locomotor ataxia. The attack may last for several days. It usually sets in with a gnawing, unpleasant sensation of the stomach, severe headache, and shortly after the patient vomits a clear, watery secretion of such acidity that the throat is irritated and made raw and sore. As mentioned, the attacks may be quite independent of food. The chronic condition of hyperacidity is more common. Digestion is usually retarded, particularly for the starches, and there are eructations of acid fluid and gastric distress. There are instances also in which when the stomach contains no food there is a secretion of a highly acid juice. In these cases burning acid eructations, or even vomiting, occurring during the night or early in the morning, are quite characteristic.

The relation of hyperacidity to gastric ulcer will be considered later.

(3) **Nervous Vomiting; Peristaltic Unrest; Rumination.**—(*a*) *Nervous Vomiting*—a condition which is not associated with anatomical changes in the stomach or with any state of the contents, but is due to nervous influences acting either directly or indirectly upon the centres presiding over the act of vomiting. The patients are, as a rule, women—

usually brunettes—and the subject of more or less marked hysterical manifestations. A special feature of this form is the absence of the preliminary nausea and of the straining efforts of the ordinary act of vomiting. It is rather a regurgitation, and without visible effort and without gagging the mouth is filled with the contents of the stomach, which are then spat out. It comes on, as a rule, after eating, but may occur at irregular intervals. In some cases the nutrition is not impaired, a feature which may give a clew to the true nature of the disease, as there may be no other hysterical manifestation present. As noted by Tuckwell, it may occur in children. Nervous vomiting is rarely serious. Death may, however, follow, as in the case reported by Garland,* in which a young woman, aged twenty, had had from the age of two attacks of vomiting which lasted for twenty-four hours, and which were very apt to occur when the child was extra well and vivacious. She had St. Vitus's dance at eleven. At about the age of twenty, she had excessive muscular twitchings, clonic in character and uncontrollable, and amounting to violent motion of the muscles. When twenty-two she had severe headache, gradually lost flesh, and became low-spirited. In January, 1884, she had headache, twitchings, and constant vomiting, and died on the 13th. There was slight atrophy of the mucous membrane of the stomach and slight increase in the firmness of the kidneys.

A type of vomiting is that associated with certain diseases of the nervous system—particularly locomotor ataxia—forming part of the gastric crises. Leyden has reported cases of primary periodic vomiting, which he regards as a neurosis.

(*b*) *Peristaltic Unrest.*—This condition, as described by Kussmaul, is an extremely common and distressing symptom in neurasthenia. Shortly after eating the peristaltic movements of the stomach are increased, and borborygmi and gurgling may be heard, even at a distance. The subjective sensations are most annoying, and it would appear as if in the hyperæsthetic condition of the nervous system the patient felt normal peristalsis, just as in these states the usual beating of the heart may be perceptible to him. A further analogy is afforded by the fact that emotion increases this peristalsis. It may extend to the intestines, particularly to the duodenum, and on palpation over this region the gurgling is most marked. The movement may be anti-peristalsis, in which the wave passes from left to right, a condition which may also extend to the intestines. There are cases on record in which colored enemata or even scybala have been discharged from the mouth.

(*c*) *Rumination; Merycismus.*—In this remarkable and rare condition the patients regurgitate and chew the cud like ruminants. It occurs in neurasthenic or hysterical persons, epileptics, and idiots. In some instances it is hereditary. There is an instance in which a governess taught

* Transactions of the Association of American Physicians, vol. iv.

it to two children. The habit may persist for years, and does not necessarily impair the health.

Treatment of Neuroses of the Stomach.—The gastralgia, if very severe, requires morphia, which is best administered subcutaneously in combination with atropia. In the milder attacks the combination of morphia (gr. $\frac{1}{8}$) with cocaine and belladonna is recommended by Ewald. The greatest caution should, however, be exercised in these cases in the use of the hypodermic syringe. It is preferable, if opium is necessary, to give it by the mouth, and not to let the patient know the character of the drug. Chloroform, in from ten to twenty drop doses, or Hoffman's anodyne will sometimes allay the severe pains. The general condition should receive careful attention, and in many cases the attacks recur until the health is restored by change of air with the prolonged use of arsenic. If there is anæmia iron may be given freely. Nitrate of silver in doses of gr. $\frac{1}{4}$ to $\frac{1}{2}$ in a large claret-glass of water taken on an empty stomach is useful in some cases.

Many cases of nervous dyspepsia with marked neurasthenic or hysterical symptoms do well on the Weir-Mitchell treatment, and in obstinate forms it should be given a thorough trial. The most striking results are perhaps seen in the cases of anorexia nervosa, which will be referred to subsequently. It is also of value in the nervous vomiting. In the distressing cases of hyperacidity, in addition to the treatment of the general neurotic condition, alkalies must be employed, either in the form of magnesia or bicarbonate of soda. The burning acid eructations are usually relieved in this way.

Limiting the patient to a strictly meat diet is a valuable procedure in many cases of dyspepsia associated with hyperacidity. The meat should be taken either raw or, if an insuperable objection exists to this, very slightly cooked. It is best given finely minced or grated on stale bread. An ample dietary is 3¼ ounces (100 grammes) of meat, two medium slices of stale bread, and an ounce (30 grammes) of butter. This may be taken three times a day with a glass of Apollinaris water, soda water, or, what is just as satisfactory, spring water. The fluid should not be taken too cold. Special care should be had in the examination of the meat to guard against tape-worm infection, but suitable instructions on this point can be given. This is sufficient for an adult man, and many obstinate cases yield satisfactorily to a month or six weeks of this treatment, after which time the less readily digested articles of food may be gradually added to the dietary. In other instances the use of the stomach-tube is most effectual.

There are forms of nervous dyspepsia occurring in women who are often well nourished and with a good color, yet who suffer—particularly at night—with flatulency and abdominal distress. The sleep may be quiet and undisturbed for two or three hours, when they are aroused with painful sensations in the abdomen and eructations. The appetite and diges-

tion may appear to be normal. Constipation is, however, usually present. In many of these patients the condition seems rather intestinal dyspepsia, and the distress is due to the accumulation of gases, the result of excessive putrefaction. The fats, starches, and sugars should be restricted. A diastase ferment is sometimes useful. The flatulency may be treated by the methods above mentioned. Naphthalin, salicylate of bismuth, and salol have been recommended. Some of these cases obtain relief from thorough irrigation of the colon at bedtime.

V. DILATATION OF THE STOMACH (*Gastrectasis*).

Etiology.—This may occur either as an acute or a chronic condition.

Acute dilatation is rarely seen, though it occurs whenever enormous quantities of food and drink are quickly ingested. Occasionally this leads to extreme paralytic dilatation, and Fagge has described two cases which came on in this way, one of which proved fatal.

Chronic dilatation results from: (*a*) Narrowing of the pylorus or of the duodenum by the cicatrization of an ulcer, hypertrophic stenosis of the pylorus (whether cancerous or simple), congenital stricture, or occasionally by pressure from without of a tumor or of a floating kidney. (*b*) Relative or absolute insufficiency of the muscular power of the stomach, due, on the one hand, to repeated overfilling of the organ with food and drink (*Ueberanstrengung des Magens*, Strümpell), and, on the other, to atony of the coats induced by chronic inflammation or degeneration or impaired nutrition, the result of constitutional affections, as cancer, tuberculosis, anæmia, etc.

The most extreme forms are met with in the first group, and most commonly as a sequence of the cicatricial contraction of an ulcer. There may be considerable stenosis without much dilatation, the obstruction being compensated by hypertrophy of the muscular coats. Considerable attention has been directed in Germany by Litten, Ewald, and others to the association of dilatation with dislocation of the right kidney. Two well-marked instances have come under my observation among a very large number of cases of movable kidney, but in neither was the dilatation extreme.

In the second group, due to atony of the muscular coats, we must distinguish between instances in which the stomach is simply enlarged and those with actual dilatation, the conditions which Ewald characterized as *megastrie* and *gastrectasis* respectively. The size of the stomach varies greatly in different individuals, and the maximum capacity of a normal organ Ewald places at about 1,600 c. c. Measurements above this point indicate absolute dilatation.

Atonic dilatation of the stomach may result from weakness of the

coats, due to repeated overdistention or to chronic catarrh of the mucous membrane, or to the general muscular debility which is associated with chronic wasting disorders of all sorts. The combination of chronic gastric catarrh with overfeeding and excessive drinking is one of the most fruitful sources of atonic dilatation, as pointed out by Naunyn. The condition is frequently seen in diabetics, in the insane, and in beer-drinkers. In Germany this form is very common in men employed in the breweries, who sometimes drink from twenty to thirty litres of beer in the day. The extraordinary size to which the organ attains in some of these cases is well shown by the *papier-maché* models which have been prepared under von Ziemssen's directions. Possibly muscular weakness of the coats may result in some cases from disturbed innervation. Dilatation of the stomach is most frequent in middle-aged or elderly persons, but the condition is not uncommon in children, especially in association with rickets.

Symptoms.—These are very variable and depend upon the cause and the degree of dilatation. Naturally the features in cancer of the pylorus would be very different from those met with in an excessive drinker. Dyspepsia is present in nearly all cases, and there are feelings of distress and uneasiness in the region of the stomach. The patient may complain much of hunger and thirst and eat and drink freely. The most characteristic symptom is the vomiting at intervals of enormous quantities of liquid and of food, amounting sometimes to four or more litres. The material is often of a dark-grayish color, with a characteristic sour odor due to the organic acids present, and contains mucus and remnants of food. On standing it separates into three layers, the lowest consisting of food, the middle of a turbid, dark-gray fluid, and the uppermost of a brownish froth. The microscopical examination shows a large variety of bacteria, yeast fungi, and the sarcina ventriculi. There may also be cherry stones, plum stones, and grape seeds.

Chemically the hydrochloric acid may be absent, diminished, normal, or in excess, depending upon the cause of the dilatation. The fermentation produces lactic, butyric, and, possibly, acetic acids and various gases.

In consequence of the small amount of fluid which passes from the stomach or is absorbed there are constipation, scanty urine, and extreme dryness of the skin. The general nutrition of the patient suffers greatly; there is loss of flesh and strength, and in some cases the most extreme emaciation. A very remarkable symptom which occurs occasionally is tetany, first described by Küssmaul. The spasm affects chiefly the muscles of the hands, arms, and legs. Loss of consciousness may occur. The spasms last for a short time only. Müller has collected eight cases of the kind, two of which occurred in simple dilatation of the stomach.

Physical Signs.—*Inspection.*—The abdomen may be large and prominent, the greatest projection occurring below the navel in the standing posture. In some instances the outline of the distended stomach can be

plainly seen, the small curvature a couple of inches below the ensiform cartilage, and the greater curvature passing obliquely from the tip of the tenth rib on the left side, toward the pubes, and then curving upward to the right costal margin. There are instances in which inspection alone reveals, at a glance, the nature of the case. Active peristalsis may be seen in the dilated organ, the waves passing from left to right. Occasionally anti-peristalsis may be seen. In cases of stricture, particularly of hypertrophic stenosis, as the peristaltic wave reaches the pylorus, the tumor-like thickening can sometimes be distinctly seen through the thin abdominal wall. To stimulate the peristalsis the abdomen may be flipped with a wet towel.

Palpation.—The peristalsis may be felt, and usually in stenosis the tumor is evident at the pylorus. The resistance of a dilated stomach is peculiar, and has been aptly compared to that of an air cushion. Bimanual palpation elicits a splashing sound, which is, of course, not distinctive, as it can be obtained whenever there is much liquid and air in the organ, but it cannot be obtained in a healthy person two or three hours after eating. The splashing may be very loud, and the patient may produce it himself by suddenly depressing the diaphragm, or it may be readily obtained by shaking him. A tube passed into the stomach may be felt externally through the skin, a procedure no longer recommended by Leube, who suggested it.

Percussion.—The note is tympanitic over the greater portion of a dilated stomach; in the dependent part the note is dull. In the upright position the percussion should be made from above downward, in the left parasternal line, until a change in resonance is reached. The line of this should be marked, and the patient examined in the recumbent position, when it will be found to have altered its level. When this is on a line with the navel or below it, dilatation of the stomach may generally be assumed to exist. This sign may be deceptive in women with lax abdomen, as the whole organ may be depressed, the lesser curvature coming, perhaps, as low as the navel. The fluid may be withdrawn from the stomach with a tube, and the dulness so made to disappear, or it may be increased by pouring in more fluid. In cases of doubt the organ may be artificially distended with carbonic-acid gas. A teaspoonful of bicarbonate of soda is first given in a little water, and then the same quantity of tartaric acid. The most accurate method of determining the size of the stomach is by inflation through a stomach-tube with a Davidson's syringe. Pacanowski has shown that the greatest vertical diameter of gastric resonance in the normal stomach varies from 10 to 14 cm. in the male and is about 10 cm. in the female.

Auscultation.—The *clapotement* or succussion can be obtained readily. Frequently a curious sizzling sound is present, not unlike that heard when the ear is placed over a soda-water bottle when first opened. It can be heard naturally, and is usually evident when the artificial gas is being

generated. The heart sounds may sometimes be transmitted with great clearness and with a metallic quality.

Mensuration may be used by passing a hard sound into the stomach until the greater curvature is reached. Normally it rarely passes more than 60 cm., measured from the teeth, but in cases of dilatation it may pass as much as 70 cm.

Diagnosis.—The diagnosis can usually be made without much difficulty by attention to these methods of examination. Curious errors, however, are on record, one of the most remarkable of which was the confounding of dilated stomach with an ovarian cyst; even after tapping and the removal of portions of food and fruit seeds, abdominal section was performed and the dilated stomach opened. The *prognosis* is bad in cases in which there is stenosis of the pylorus, either simple or cancerous.

Treatment.—With care, the dilatation consequent upon simple stenosis is not incompatible with many years of life. In the cases due to atony careful regulation of the diet and proper treatment of the associated catarrh will suffice to effect a cure. Strychnine, ergot, and iron are recommended. Washing out the stomach is of great service, though we do not see such striking and immediate results in this form. In cases of mechanical obstruction the stomach should be emptied and thoroughly washed, either with warm water or with an antiseptic solution. As Welch states, in his exhaustive article on this subject, we accomplish in this way three important things: We remove the weight, which helps to distend the organ; we remove the mucus and the stagnating and fermenting material which irritates and inflames the stomach and impedes digestion; and we cleanse the inner surface of the organ by the application of water and medicinal substances. The introduction of this method by Küssmaul, in 1867, has practically revolutionized the treatment in diseases of the stomach. The method of application has already been referred to. The patient can usually be taught to wash out his own stomach, and in a case of dilatation from simple stricture I have known the practice to be followed daily for three years with great benefit. The rapid reduction in the size of the stomach is often remarkable, the vomiting ceases, the food is taken readily, and in many cases the general nutrition improves rapidly. As a rule, once a day is sufficient, and it may be practised either the first thing in the morning or before going to bed. So soon as the fermentative processes have been checked, lukewarm water alone should be used.

The food should be taken in small quantities at frequent intervals, and should consist of scraped beef, Leube's beef solution, and tender meats of all sorts. Fatty and starchy articles of diet are to be avoided. Liquids should be taken sparingly.

In cicatricial stenosis of the pylorus Loreta has practised dilatation with considerable success. The statistics of Barton show that of 25 published cases 15 recovered and 10 died.

VI. THE PEPTIC ULCER—GASTRIC AND DUODENAL.

The round, perforating or simple ulcer is usually single and occurs in the stomach and in the duodenum as far as the papilla biliaria. It probably follows nutritional disturbance in a limited region of the mucosa, which results in the gradual destruction of this area by the gastric juice. The condition is usually associated with hyperacidity.

Etiology.—Clinically the simple ulcer is not so frequent as the statistics of post-mortems would lead us to expect; thus in the extensive records collected by Welch, ulcer, cicatrized or open, was present in about five per cent of persons dying from all causes. The scars are found more frequently than the open ulcer.

Females are more frequently affected than males. Of 1,699 cases collected from hospital statistics by Welch, and examined post mortem, 40 per cent were in males and 60 per cent were in females. He gives the age incidence in 607 cases, of which three fourths were distributed between the ages of twenty and sixty, with tolerable uniformity in the four decades. In females the largest number of cases occurs between twenty and thirty; in males, between thirty and forty. Ulcer occasionally occurs in children, and Goodhart has reported a case in an infant thirty hours old. Gastric ulcer is stated to be less common in this country than in Europe.

In women it is frequent among servant girls, and in men who follow such occupations as shoe-making, weaving, and tailoring, possibly connected, as Habershon suggested, with pressure on the stomach. This view has been developed by Rasmussen, who holds that pressure of the costal margin, from various causes, induces anæmia and atrophy of the mucous membrane, particularly in the region of the smaller curvature. Very rarely the disease originates from traumatism or the action of corrosive fluids. Gastric ulcer is associated in a special manner with certain diseases, in women with anæmia and chlorosis and with menstrual disorders. It is not infrequently met with in tuberculosis. Such cases are not, however, to be mistaken for the true tuberculous ulcer, which may be found in the stomach.

Many cases have occurred in connection with disease of the heart or of the blood-vessels, a relation of special interest in connection with the embolic theory of its production.

The duodenal ulcer is less common than the gastric ulcer, and occurs most frequently in males. The combined statistics of Krauss, Chvostek, Lebert, and Trier give 171 cases in males and 39 in females. In 9 cases which have come under my observation 7 were in males and 2 in females; one of these was in a lad of twelve. It has been found in association with tuberculosis, and may follow large superficial burns.

Morbid Anatomy.—Though usually single, the ulcers may be multiple. In none of my cases were there more than five, but there is an instance

on record of thirty-four. The ulcer is situated most commonly on the posterior wall of the pyloric portion at or near the lesser curvature. It is not nearly so frequent on the anterior wall. Of 793 cases collected by Welch from hospital statistics, 288 were on the lesser curvature, 235 on the posterior wall, 95 at the pylorus, 69 on the anterior wall, 50 at the cardia, 29 at the fundus, 27 on the greater curvature. The duodenal ulcer is usually situated just outside the ring in the first portion of the gut.

The ulcer varies from 1 to 10 cm. in diameter. It may be small and punched out, or it may reach an enormous size. The largest of which I have any knowledge is one reported by Peabody, which measured 19 by 10 cm. and involved all of the lesser curvature and spread over a large part of the anterior and posterior walls. The ulcer is usually round or oval in shape, but may be irregular with sinuous borders. It is often distinctly terraced. In acute cases the mucous membrane is sharply cut, as if punched out by an instrument. In old cases the edge is indurated and loses the sharp margin. The floor is formed either by the submucosa, by the muscular layers, or, not infrequently, by the neighboring organs, to which the stomach has become attached. In the healing of the ulcer, if the mucosa is alone involved, the granulation tissue develops from the edges and the floor and the newly formed tissue gradually contracts and unites the margins, leaving a smooth scar. In larger ulcers which have become deep and involved the muscular coat the cicatricial contraction may cause serious changes, the most important of which is narrowing of the pyloric orifice and consequent dilatation of the stomach. In the case of a girdle ulcer, hour-glass contraction of the stomach may be produced. It is probable that large ulcers persist for years without any attempt at healing.

The ulcer may deepen and penetrate the coats. Fortunately, in a majority of the cases, adhesions form between the stomach and adjacent organs, particularly with the pancreas, the left lobe of the liver, and the omental tissues On the anterior surface of the stomach adhesions do not so readily form, hence the great danger of the ulcer in this situation, which more readily perforates and excites a diffuse and fatal peritonitis. On the posterior wall the ulcer penetrates directly into the lesser peritoneal cavity, in which case it may produce an . ır-containing abscess with the symptoms of the condition known as subphrenic pyo-pneumothorax. In rare instances adhesions and a gastro-cutaneous fistula form, usually in the umbilical region. Fistulous communication with the colon may also occur, or a gastro-duodenal fistula. There are several instances on record of perforation into the pericardium, and at least two of rupture into the left ventricle. Perforation into the pleura may also occur. It is to be noted that general emphysema of the subcutaneous tissues occasionally follows perforation of a gastric ulcer.

One of the most serious effects of gastric ulcer is erosion of blood-vessels. The hæmorrhage may occur in the acutely formed ulcer or in the

ulceration which takes place at the base of the chronic form; it is in the latter condition that the bleeding is most common. Ulcers on the posterior wall may erode the splenic artery, but perhaps more frequently the bleeding proceeds from the artery of the lesser curve. In the case of duodenal ulcer the pancreatico-duodenal artery may be eroded or (as in one of my cases) fatal hæmorrhage may result from the opening of the hepatic artery, or more rarely the portal vein. Interesting changes occur in the vessels. Embolism of the artery supplying the ulcerated region has been met with in several cases; in others diffuse endarteritis. Small aneurisms have been found in the floor of the ulcers by Douglas Powell, Welch, and others.

The mode of the origin of the peptic ulcer has been much discussed. Ulcers have been produced in animals in many ways, both by artificial emboli and by direct chemical and mechanical irritants applied to the mucosa. The ulcers thus produced heal with great rapidity unless the animals have been rendered anæmic by repeated abstraction of blood. Virchow's view that the process may result from plugging the nutrient artery of the part, either by an embolus or by a thrombus, and the infarct so produced is destroyed by the gastric juice, has gained general acceptance. It is in conformity with Pavy's well-known experiments and with the anatomical facts already mentioned, particularly with the funnel-like shape of the ulcer, and the actual demonstration, in some cases, of the plugged vessels; but this view scarcely meets all the cases, in many of which the etiology is still obscure. Mere mechanical injury to the mucous membrane is, however, in most cases, insufficient cause for an ulcer, for normally the stomach is perfectly able to withstand such insults. Ewald concludes that certain predisposing causes play an important *rôle* in its development. He points to its frequency in conditions of amenorrhœa, chlorosis, anæmia after confinements, etc., where one may assume that the condition of the blood is not wholly normal, and also to the fact that in the majority of cases of this affection there is a hyperacidity of the gastric juice. One or both of these predisposing factors seem to be present in most cases, and it has been recently shown that in the various anæmiæ there is an appreciable diminution in the normal alkalinity of the blood, a fact which tends to explain one of the predisposing causes in these affections, and which is in accord with the "alkalescence theory" of Cohnheim *et al.* The duodenal ulcer has an identical origin, but a few cases of acute ulcer, as already mentioned, have a curious relation with superficial burns. In one of my cases there was an ulcer in the posterior wall of the duodenum, 1·5 cm. in diameter, with overlapping edges, and not far from it was a cyst-like cavity in the submucosa associated with Brunner's glands, and it is possible that the open ulcer, with undermined edges, resulted from the rupture of one of these cysts.

Symptoms.—The condition may be met with accidentally, post mortem, in cases which have presented no indication of gastric disturbance.

In other instances the first symptoms may be due to perforation. In others again the symptoms, for months and years, may be those of ordinary dyspepsia, and the ulcer may not have been suspected until the occurrence perhaps of a sudden hæmorrhage.

The symptoms suggestive of peptic ulcer are: (*a*) Dyspepsia, which may be slight and trifling or of a most aggravated character. In a considerable proportion of all cases nausea and vomiting occur, the latter not for two or more hours after eating. The vomitus usually contains a large amount of HCl.

(*b*) Hæmorrhage is present in at least one half of all cases. It may be slight, but more commonly is profuse, and may be in such quantities and brought up so quickly that it is fluid, bright red in color, and quite unaltered. When the blood remains for some time in the stomach and is mixed with food it may be greatly changed, but the vomiting of a large quantity of unaltered blood is very characteristic of ulcer. Syncope may follow or death may directly result from the hæmorrhage. A most extreme grade of anæmia may be produced. In either the gastric or duodenal ulcer, more commonly in the latter, the blood may be passed in the stools and not be vomited. This may occur when the hæmorrhage is slight, but also when it is profuse enough to produce collapse and extreme anæmia.

(*c*) Pain is perhaps the most constant and distinctive feature of ulcer. It varies greatly in character; it may be only a gnawing or burning sensation, which is particularly felt when the stomach is empty, and is relieved by taking food, but the more characteristic form comes on in paroxysms of the most intense gastralgia, in which the pain is not only felt in the epigastrium, but radiates to the back and to the sides. These attacks are most frequently induced by taking food, and they may recur at a variable period after eating, sometimes within fifteen or twenty minutes, at others as late as two or three hours. It is usually stated that when the ulcer is near the cardia the pain is apt to set in earlier, but there is no certainty on this point. The attacks may occur at intervals with great intensity for weeks or months at a time, so that the patient constantly requires morphia, then again they may disappear entirely for a prolonged period. In the attack the patient is usually bent forward, and finds relief from pressure in the epigastric region; one patient during the attack would lean over the back of a chair; another would lie flat on the floor, with a hard pillow under the abdomen. Pressure is, as a rule, grateful. It has been thought that the posture assumed during the attack would indicate the site of the ulcer, but this is very doubtful.

(*d*) Tenderness on pressure is a common symptom in ulcer, and patients wear the waist-band very low. There may be a painful point of very limited extent, most frequently an inch or two below the ensiform cartilage. In old ulcers with thickened bases an indurated mass can usually be felt in the neighborhood of the pylorus. Pressure should be made

with great care, as rupture of an ulcer has been induced by careless manipulation.

(*e*) Of general symptoms, loss of weight results from the prolonged dyspepsia, but it rarely, except in association with cicatricial stenosis of the pylorus, reaches the high grade met with in cancer. The anæmia may be extreme, and in one case of duodenal ulcer which I examined the blood count was as low as 700,000 per c. mm. There are instances, such as the one reported by Pepper and Griffith, in which the extreme anæmia cannot be explained by the occurrence of hæmorrhage.

According to Welch, perforation occurs in about six and a half per cent of all cases. The acute, perforating form is much more common in women than in men. The symptoms are those of perforative peritonitis. In some instances the pain associated with perforation is not referred to the abdomen. In a case of H. C. Wood's the chief symptoms were pain in the left shoulder and excessive pain in the back on movement. Perforation is not necessarily fatal. Several cases of recovery have been reported.

The course of the disease is, in the majority of cases, chronic. Only a few instances run a very acute course. The following group of clinical forms, described by Welch, indicate the diversity of this affection:

"1. Latent ulcers, with entire absence of symptoms, and revealed as open ulcers or as cicatrices at the autopsy.

"2. Acute perforating ulcers. With or without a period of brief gastric disturbance, perforation occurs and causes speedy death.

"3. Acute hæmorrhagic form of gastric ulcer. After a latent or a brief course of the ulcer, profuse gastrorrhagia occurs, which may terminate fatally or may be followed by the symptoms of chronic ulcer.

"4. Gastralgic-dyspeptic form. In this, which is the most common form of gastric ulcer, gastralgia, dyspepsia, and vomiting are the symptoms. Sometimes one of the symptoms predominates greatly over the others, so that Lebert distinguishes separately a gastralgic, a dyspeptic, and a vomitive variety. Gastralgia is the most frequent symptom.

"5. Chronic hæmorrhagic form. Gastrorrhagia is a marked symptom, and occurs usually in combination with the symptoms just mentioned.

"6. Cachectic form. This usually corresponds only to the final stage of one of the preceding forms, but the cachexia may develop so rapidly and become so marked that the course of the disease closely resembles that of gastric cancer.

"7. Recurrent form. In this the symptoms of gastric ulcer disappear, and then follow intervals, often of considerable duration, in which there is apparent cure, but the symptoms return, especially after some indiscretion in the mode of living. This intermittent course may continue for many years. In these cases it is probable either that fresh ulcers form or that the cicatrix of an old ulcer becomes ulcerated.

"8. Stenotic form. By the formation of cicatricial tissue in and

around the ulcer, the pyloric orifice becomes obstructed and the symptoms of dilatation of the stomach develop."

The course may be very protracted, and there are cases in which the disease has persisted for over twenty years. I have reported two instances of peptic ulcer, probably duodenal, in which well-marked symptoms were present, in one case for eighteen, and in the other for twelve years. Both were of the chronic hæmorrhagic form.

Diagnosis.—The recognition of gastric ulcer is in many cases easy, as the combination of dyspepsia, gastralgic attacks, and hæmatemesis is very characteristic. Of the symptoms, hæmorrhage with the gastralgic attack is the most characteristic. The distinctions between ulcer and cancer will be given. The greatest difficulty is offered by certain cases of gastralgia, which may resemble ulcer very closely, as, with the exception of the hæmorrhage, there is no single symptom which may not be present. Even with hæmorrhage the case may not be clear, and no less an authority than the late Austin Flint made a diagnosis of recurring gastralgia in a patient who had, on and off for nine years, violent pains with vomiting in association with ulcer. A difficulty also results from the fact that in many instances gastralgia is one of the symtoms of nervous dyspepsia, and may exist with marked emaciation.

The following points are of value in discriminating between these two conditions:

(*a*) In ulcer the pain is more definitely connected with taking food, though this is not always the case, as in the duodenal form the gastralgic attacks may occur at night when the stomach is empty. Relief of pain after eating is certainly less common in ulcer than in gastralgia, though it is a very uncertain feature, and in certain cases the pain in ulcer is *always* relieved by taking food.

(*b*) In ulcer dyspeptic symptoms are almost invariably present in the intervals between the attacks, and even when pain is absent there is slight distress.

(*c*) Local sensitiveness in a particular spot in the epigastrium is suggestive of ulcer. External pressure usually aggravates the pain in ulcer, and often relieves it in gastralgia. This is, however, a very uncertain feature, as patients writhing with the pains of ulcer may press the abdomen over the back of a chair or place a hard pillow under it.

(*d*) The general condition and history of the patient often give the most trustworthy information. The nutrition is impaired more frequently in ulcer than in gastralgia. In the former we find more commonly (in women) dysmenorrhœa and chlorosis, while in the latter there are associated nervous phenomena—hysterical manifestations or neuralgias in other regions.

(*e*) On examination of the abdomen, not only is pain on pressure much more common in ulcer, but there may also be thickening about the pylorus and, in many cases, signs of dilatation of the stomach.

(*f*) Hyperacidity of the gastric juice exists with ulcer.

The *gastric crises* which occur in affections of the spinal cord, particularly in locomotor ataxia, may simulate very closely the gastralgic attacks of ulcer, and as they so often exist in the preataxic stage their true nature may be overlooked; but the occurrence of lightning pains, the ocular symptoms, and the absence of the knee reflex are indications usually sufficient to render the diagnosis clear.

Can the gastric and duodenal ulcer be distinguished clinically? As already stated, they originate in the same way and present the same anatomical characters. In the great majority of cases they cannot be separated during life, as the symptoms produced are identical. Bucquoy has suggested that the duodenal ulcer can be distinguished by the following definite characters: (*a*) Sudden intestinal hæmorrhage in an apparently healthy person, which tends to recur and produce a profound anæmia. Hæmorrhage from the stomach may precede or accompany the melæna. (*b*) Pain in the right hypochondriac region, coming on two or three hours after eating. (*c*) Gastric crises of extreme violence, during which the hæmorrhage is more apt to occur. Certainly the occurrence of sudden intestinal hæmorrhage with gastralgic attacks is extremely suggestive of duodenal ulcer. W. W. Johnston has reported an instance in which he made the diagnosis on these symptoms, and in one of the Montreal cases Palmer Howard suggested correctly the presence of a duodenal ulcer on similar grounds. A patient under my care who had, during eighteen years, frequent attacks of hæmatemesis with gastralgia had melæna repeatedly without vomiting blood;* but as a rule in the attacks the blood was vomited first, and did not appear in the stools until later. Occasionally this symptom will be found an important aid in diagnosis. The situation of the pain is too uncertain a factor on which to lay much stress, and the character of the crises is usually identical.

Gall-stone colic may occasionally simulate the pains of gastric ulcer. The sudden onset and as sudden termination, the swelling and tenderness of the liver, the enlargement of the gall-bladder, if present, and the occurrence of jaundice are points which usually make the diagnosis clear.

Treatment.—Post-mortem observations show that a very large number of ulcers heal completely, but the process is slow and tedious, often requiring months, or, in severe cases, years. The following are the important points in treatment:

(*a*) Absolute rest in bed.

(*b*) A carefully and systematically regulated diet. While theoretically it is better to give the stomach complete rest by rectal feeding, yet in practice this strict limitation is not found satisfactory. The food should be bland, easily digested, and given at stated intervals. The following dietary will be found useful: At 8 A. M. give 200 c. c. of Leube's beef solu-

* On the Diagnosis of Duodenal Ulcer, Medical Record, November 24, 1888.

tion; at 12 M., 300 c. c. of milk gruel or peptonized milk. The gruel should be made with ordinary flour or arrowroot, and is mixed with an equal quantity of milk. If necessary it may be peptonized. Buttermilk is very well borne by these patients. At 4 P. M. the beef solution again, and at 8 P. M. the milk gruel or the buttermilk.

The stomach in some cases is so irritable that the smallest amount of food is not well borne. In such cases lavage may be practised, if necessary, every morning and evening, with mildly alkaline water, after which the beef solution is given and the feeding supplemented by the rectal injections. Ill effects rarely follow the careful use of the stomach tube in gastric ulcer. There are some cases which do well from the outset on a milk diet, given at regular intervals, three or four ounces every two hours. When milk is not well borne egg albumen may be substituted, or the whites of eight eggs may be alternated with Leube's beef solution. At the end of a month, if the condition has improved, the patient may be allowed scraped beef or young chicken, perfectly fresh sweet-bread, and farinaceous puddings made with milk and eggs. Local applications, such as warm fomentations, over the abdomen are very useful. The patient should be told that the treatment will take at least three months, and for the greater portion of the time he should be in bed.

(*c*) Medicinal measures are of very little value in gastric ulcer, and the remedies employed do not probably benefit the ulcer, but the gastric catarrh. The Carlsbad salts are warmly recommended by von Ziemssen. The artificial preparation (sulphate of sodium, 50; bicarbonate of sodium, 6; chloride of sodium, 3) may be substituted, of which a teaspoonful is taken every morning. Bismuth, in doses of thirty to sixty grains three times a day, and nitrate of silver may be given, but they influence the associated conditions rather than the ulcer.

The pain if severe requires opium. Unless the gastralgia is intense morphia should not be given hypodermically, as there is a very serious danger in these cases of establishing the morphia habit. Doses of an eighth of a grain, with the bicarbonate of soda and bismuth, will allay the mild attacks, but the very severe ones require the hypodermic injection of a quarter or often half a grain. Antipyrin and antifebrin may be tried, but, as a rule, are quite ineffectual. In the milder attacks Hoffman's anodyne, or twenty or thirty drops of chloroform, or the spirits of camphor will give relief. Counter-irritation over the stomach with mustard or cantharides is often useful.

For the vomiting there is no measure so successful as lavage. If intractable the patient must be fed per rectum. The patient will sometimes retain food which is passed into the stomach through the tube, and Leube's beef solution or milk may be given in this way. Cracked ice, chloroform, oxalate of cerium, bismuth, hydrocyanic acid, and ingluvin may be tried. When hæmorrhage occurs the patient should be put under the influence of opium as rapidly as possible. No attempt should be made

to check the hæmorrhage by administering medicines through the mouth; as the profuse bleeding is always from an eroded artery, frequently from one of considerable size, it is doubtful if acetate of lead, tannic and gallic acids, and the usual remedies have the slightest influence. The essential point is to give rest, which is best obtained by opium. Ergotin may be administered hypodermically in two-grain doses. Nothing should be given by the mouth except small quantities of ice. In profuse bleeding a ligature may be applied around a leg, or a leg and arm. Not infrequently the loss of blood is so great that the patient faints. A fatal result is not, however, very common from hæmorrhage. Transfusion may be necessary, or, still better, the subcutaneous infusion of saline solution.

The patients usually recover rapidly from the hæmorrhage and require iron in full doses, which may, if necessary, be given hypodermically.

VII. CANCER OF THE STOMACH.

Etiology.—The stomach comes next to the uterus as the most frequent seat of primary cancer, amounting, as shown by the statistics of Welch,* to 21·4 per cent in a total of over 30,000 cases. The ratio of males to females affected is about five to four. Age has an important bearing. Of 2,038 cases tabulated by this author three fourths occurred between the fortieth and the seventieth year, 24·5 per cent between the ages of forty and fifty, and 30·4 between the ages of fifty and sixty. In childhood it is extremely rare. Cancer of the stomach is a very common disease in this country, though statistics would indicate that it is rather less frequent than in Europe. With reference to heredity, Welch analyzed 1,744 cases and found that a family history was present in 243. Local conditions, such as chronic gastritis and traumatism, have been thought by some to be important factors. Cancer may develop in a simple ulcer of the stomach, but this sequence is extremely rare. It is not probable that depressing emotions, mode of life, or previous disease have any influence whatever in the causation of cancer.

Morbid Anatomy.—The most common varieties of gastric cancer are the cylindrical-celled epithelioma and the encephaloid; next in frequency is scirrhous, and then colloid cancer. With reference to the situation of the tumor, Welch analyzed 1,300 cases, in which the distribution was as follows: Pyloric region, 791; lesser curvature, 148; cardia, 104; posterior wall, 68; the whole or greater part of the stomach, 61; multiple tumors, 45; greater curvature, 34; anterior wall, 30; fundus, 19.

The medullary cancer occurs in soft masses, which involve all the coats of the stomach and usually ulcerate early. The tumor may form villous projections or cauliflower-like outgrowths. It is soft, grayish white in color,

* System of Medicine, vol. ii, Philadelphia, 1886.

and contains much blood. Microscopically it shows a scanty stroma, enclosing alveoli which contain irregular polyhedral and cylindrical cells. The cylindrical-celled epithelioma may also form large irregular masses, but the consistence is usually firmer, particularly at the edges of the cancerous ulcers. Microscopically the section shows elongated tubular spaces filled with columnar epithelium, and the intervening stroma is abundant. Cysts are not uncommon in this form. The scirrhous variety is characterized by great hardness, due to the abundance of the stroma and the limited amount of alveolar structures. It is seen most frequently at the pylorus, where it is a common cause of stenosis. It may be combined with the medullary form. The colloid cancer is peculiar in its wide-spread invasion of all the coats. It also spreads with greater frequency to the neighboring parts, and it occasionally causes extensive secondary growths of the same nature in other organs. The appearance on section is very distinctive, and even with the naked eye large alveoli can be seen filled with the translucent colloid material. The term alveolar cancer is often applied to this form. Ulceration is not constantly present, and there are instances in which, with most extensive disease, digestion has been very slightly disturbed. There is a specimen in the Warren Museum, at the Harvard Medical School, of the most wide-spread colloid cancer, in which the stomach contained after death large portions of undigested beef-steak.

Secondary cancer may also occur in the stomach. Welch has collected 37 cases, 17 of which were secondary to cancer of the breast. The cancer may produce important changes in the position and shape of the organ, particularly when the orifices are involved; thus, a cancer at the cardia may be associated with wasting of the organ and reduction in its size. The œsophagus above the obstruction may be greatly distended. On the other hand, annular cancer at the pylorus may cause stenosis and great dilatation of the organ; not necessarily, however, as there are instances on record in which the pylorus has been extremely narrowed without any increase in the size of the stomach. In scirrhous cancer the organ may be very greatly thickened and contracted. The stomach may be displaced or altered in shape by the weight of the tumor, particularly in cancer of the pylorus, which has been found in every region of the abdomen, and even in the true pelvis. The mobility of the tumors is at times extraordinary and very deceptive. There was in the Philadelphia Hospital an old man with a tumor at the pylorus the size of a cricket ball, which was usually in the epigastric region, but could be pushed into the right hypochondria or into the splenic region entirely beneath the ribs. Adhesions very frequently occur, particularly to the colon, the liver, and the anterior abdominal wall.

Secondary cancerous growths are very frequent, as shown by the following analysis by Welch of 1,574 cases: Metastasis occurred in the lymphatic glands in 551; in the liver in 475; in the peritonæum, omentum, and intestine in 357; in the pancreas in 122; in the pleura and lung in

98; in the spleen in 26; in the brain and meninges in 9; in other parts in 92. The lymph glands affected are usually those of the abdomen, but the cervical and inguinal glands are not infrequently attacked, and give an important clew in diagnosis. Occasionally, a secondary metastatic growth occurs subcutaneously, either at the navel or beneath the skin in the vicinity. In an instance recently under observation in a patient with jaundice, which developed somewhat suddenly and was believed to be catarrhal, there were no signs of enlargement of the liver or tumor of the stomach, but a nodular body developed at the navel, which on removal proved to be typical scirrhus. A second case in the ward at the same time, with an obscure doubtful tumor in the left hypochondria, developed a painful nodular subcutaneous growth midway between the navel and the left margin of the ribs.

In the extensive ulceration which occurs perforation of the stomach is not uncommon. It occurred into the peritonæum in 17 of the 507 cases of cancer of the stomach collected by Brinton. When adhesions form, the most extensive destruction of the walls may take place without perforation into the peritoneal cavity. In one instance which came under my observation a large portion of the left lobe of the liver lay within the stomach. Occasionally a gastro-cutaneous fistula is established. Perforation may occur into the colon, the small bowel, the pleura, the lung, or into the pericardium.

Symptoms.—Cancer of the stomach may not produce symptoms other than gradual failure of health, and death may take place from asthenia without any suspicion of the existence of malignant disease. These cases are not uncommon, particularly in elderly persons in institutions. In a great majority of all cases there are very definite symptoms, but the disease presents a very diverse clinical picture. Certain general features stand out with special prominence. The onset is insidious, sometimes with gastric disturbance, but more commonly with impairment of health and strength. A dyspepsia which may have been troublesome for years becomes aggravated. Ewald, however, states that dyspeptic symptoms are rare prior to the onset of gastric cancer. There are attacks of nausea and vomiting, and there is pain in the region of the stomach, which is aggravated by taking food. The patient emaciates, the anæmia becomes pronounced, and the prostration may be extreme. With slight intermissions the course is progressively downward, and from month to month the loss is striking. The face has a sallow cachectic appearance, the anæmia becomes more intense, and there may be œdema of the ankles. Blood may be present in the vomited matter. If with these general features a tumor can be felt in the region of the stomach the diagnosis is rendered certain. The course, in rapid cases, may be from three to six months, but as a rule the disease extends from eighteen months to two years.

Dyspepsia is common at the outset, but in so many cases the patients

have had indigestion for years that the trouble is supposed at first to be only an aggravation of the chronic complaint. Loss of the desire for food is a very frequent symptom. There are exceptional instances, however, in which the appetite is retained throughout, and the functions of the stomach very slightly disturbed. Nausea is a striking feature in many cases, and is much more common than in ulcer. There may even be a sudden repulsion at the sight of food.

Vomiting, which is one of the most constant symptoms of cancer of the stomach, may come on early, or only after the dyspepsia has persisted for some time. At first it is at long intervals, but subsequently it is more frequent, and may recur several times in the day. There are cases in which it comes on in paroxysms and then subsides; in other cases, it sets in early, persists with great violence, and may cause a fatal termination within a few weeks. Vomiting is more frequent when the cancer involves the orifices, particularly the pylorus, in which case it is usually delayed for an hour or more after taking the food. When the cardiac orifice is involved it may follow at a shorter interval. Extensive disease of the fundus or of the anterior or posterior wall may be present without the occurrence of vomiting. The vomited matters consist of food and mucus in a grayish or dark sour-smelling fluid. The food is sometimes very little changed, even after it has remained in the stomach for twenty-four hours.

Hæmorrhage is a frequent symptom, but the bleeding is rarely profuse; more commonly there is slight oozing, and the blood is mixed with, or altered by the secretions, and when vomited the material is dark brown or black, the so-called "coffee-ground" vomit. This is present in a considerable proportion of all cases of cancer, and is an important indication. The blood can be recognized by the microscope as shells of the red blood-corpuscles and irregular masses of altered blood pigment. In cases of doubt the spectroscope may be employed or hæmin crystals obtained.

Fragments of the tumor are rarely found in the vomit, and of the numerous specimens which I have had occasion to examine I have never been able to satisfy myself of the existence of cancerous tissue. As Rosenbach states, in the material washed out with the stomach-tube undoubted fragments may be found. The yeast fungus, various bacteria, and the sarcina ventriculi may be present, the latter not so often in cancer as in dilatation.

Great stress has been laid of late years upon the absence of free hydrochloric acid in the secretions. As an outcome of the enormous number of observations which have recently been made it may be said that free hydrochloric acid is absent in a majority of cases of cancer of the stomach. This defect is associated with impairment of the secreting function of the organ. The examination should be made repeatedly, by the methods already referred to, and with our present knowledge the persistent absence of free HCl in the stomach contents, taken in conjunc-

tion with other symptoms, may be regarded as highly suggestive of cancer. Unfortunately, the free acid may be absent in certain other conditions, such as atrophy, and occasionally in chronic gastritis, so that it is of greater value from the negative standpoint. As Kinnicutt expresses it, "the presence of free HCl in the stomach contents in repeated examinations in doubtful cases is of the greatest diagnostic value, and points very certainly to absence of cancer." Rosenheim has recently shown that in cases in which cancer develops in the base of an old ulcer HCl may be present throughout the course.

Pain is an early and important symptom. It is very variable in situation, and while most common in the epigastrium, it may be referred to the shoulders, the back, or the loins. The pain is described as dragging, burning, or gnawing in character, and very rarely occurs in severe paroxysms of gastralgia, as in gastric ulcer. As a rule, the pain is aggravated by taking food. There is usually marked tenderness on pressure in the epigastric region. It is, however, remarkable how many cases run a painless course.

The *physical examination* of the abdomen reveals in many instances the presence of a tumor. Inspection may show a nodular mass in the epigastrium, or the outlines of a dilated stomach, with peristaltic action. In the palpation of the stomach it is important to bear in mind certain anatomical points. At least two thirds of the organ lie in the left hypochondrium beneath the ribs, and so are practically out of reach. The pyloric orifice lies to the right of the median line, particularly when the stomach is full, in which case it may be reached. It is about on a level with the inner extremity of the eighth right costal cartilage. The pylorus is movable and changes considerably in position with the distention of the stomach. Practically, in health there is available for palpation only a part of the anterior surface of the stomach and the pylorus, which is sometimes, but not always, overlapped by the liver. Tumors limited to the cardia, even when extensive, cannot be felt at all. Tumors involving the fundus, the posterior wall, and the greater part of the lesser curvature cannot be detected unless very large. Tumors of the pylorus, of the anterior wall, and of a large part of the greater curvature are in accessible situations. In the examination the knees should be drawn up, and the patient asked to relax the abdominal walls as much as possible. Sometimes, when nothing can be felt on quiet breathing, a deep inspiration will force down the stomach and bring a tumor mass within reach. Examination should also be made in the knee-elbow position. Cancerous tumors of the stomach are usually felt in the epigastric region, but a mass at the pylorus may be felt in the umbilical region, or, in cases of extreme mobility, in a hypochondriac region, or, very exceptionally, low down in the iliac region. The tumor is usually firm, hard, nodular, and painful on pressure. At the pylorus the mass may be rounded, ball-like, and readily grasped. Gas may sometimes be felt

bubbling through it. Communicated pulsation from the aorta is not at all uncommon. Inflation of the stomach with gas is often a valuable aid in diagnosis. A teaspoonful of bicarbonate of soda is first given in water, followed by the same amount of tartaric acid. The distention of the stomach which follows may suffice to bring tumor masses into reach.

Careful examination should be made to determine the presence of secondary cancer of the liver or involvement of the lymph glands in the groins or in the supraclavicular spaces. As already mentioned, the development of nodules about the navel may give an important hint, or there may be signs of secondary involvement of the peritonæum.

Intestinal symptoms are not very common. Constipation is more frequently present than diarrhœa, which may, however, set in and prove obstinate toward the end. When there is much bleeding the stools may be dark in color.

A progressive *anæmia* is one of the most striking features of gastric cancer. As a rule the blood-count does not fall below fifty per cent. A leucocytosis is almost constantly present, and Welch has noted an instance in which the ratio of white to red corpuscles was one to twenty. There are instances in which the clinical picture is rather that of a pernicious anæmia, with reduction of the red blood-corpuscles to twenty-five per cent and marked poikilocytosis. When any degree of anæmia is present nucleated red corpuscles may be found in dried and stained specimens, and this method of examination may be of much service when an actual blood-count is impossible. The condition is, however, an anæmia with wasting, and the layer of panniculus is not retained as in the ordinary forms of pernicious anæmia. Ultimately the patient develops an aspect to which the term cachectic is applied, and which is perhaps more marked in gastric cancer than in any other disease. There may be a slight yellowish tint to the skin, and it is not uncommon to see brownish stains, the cachectic chloasma.

Associated with the anæmia and directly dependent upon it are the dropsical symptoms so common in this affection. Œdema of the ankles and of the legs is present and may progress to a general anasarca; the cases may be mistaken for heart-disease or dropsy. There are no special cardiac symptoms; the pulse becomes rapid and feeble toward the end. The anæmia may, however, produce such palpitation and dyspnœa that the case may be regarded as cardiac. Thrombosis of a femoral vein may occur.

The urine may contain a trace of albumen and, toward the close, tube-casts. Indican is often present in increased quantity, and occasionally acetone and diacetic acid.

The temperature is usually normal, and toward the end, when cachexia is well marked, subnormal. There are, however, interesting paroxysmal elevations of temperature, definite chills with fever, in which the thermometer registers 103° or 104°, followed by profuse sweating. The rigors

may recur at intervals for weeks, and, if no tumor is felt, may complicate the diagnosis. In a case at the Philadelphia Hospital the paroxysms recurred for more than six weeks. The autopsy showed a cancer of the stomach with adhesions to the colon and extensive suppuration at the base of the cancer and in a pocket between the stomach and omentum.

The mind usually remains clear to the close. Naturally the patient has attacks of despondency. Toward the close delirium is common. A form of coma resembling that which occurs in diabetes is occasionally met with in gastric cancer. The patient becomes restless or excited, and gradually unconsciousness supervenes, with or without dyspnœa. It is due to the presence of some toxic agent in the blood, possibly the diacetic acid.

Among symptoms referable to the development of secondary growths those pertaining to the liver are most important. Jaundice is not uncommon, and there may be signs of great enlargement of the liver. Many instances which are clinically recorded as primary cancer of this organ are in reality secondary to latent cancer of the stomach. The importance of enlargement of the supra-clavicular and inguinal glands in gastric cancer has already been emphasized. The new growths may extend to the peritonæum and, if there is much effusion, produce ascites. Reference has been made to the perforations liable to occur in gastric cancer. The course of the disease is progressively downward. In the majority of all cases death occurs within two years, and the average duration is not more than eighteen months. In cases of scirrhus the progress is slower.

Diagnosis.—When a tumor is present there is not much difficulty in determining the nature of the trouble; even in its absence the progressive emaciation, the loss of energy and strength, the anæmia and cachexia, when associated with marked gastric symptoms, are almost pathognomonic. There are many instances, however, in which a positive diagnosis is impossible. The diseases with which cancer is most liable to be confounded are ulcer and chronic gastric catarrh, and the differential features are so well drawn in the elaborate article by my colleague Welch that I here append them:*

GASTRIC CANCER.	GASTRIC ULCER.	CHRONIC CATARRHAL GASTRITIS.
1. Tumor is present in three fourths of the cases.	1. Tumor rare.	1. No tumor.
2. Rare under forty years of age.	2. May occur at any age after childhood. Over one half of the cases under forty years of age.	2. May occur at any age.

* *Op. cit.*, vol. ii, p. 570.

GASTRIC CANCER.	GASTRIC ULCER.	CHRONIC CATARRHAL GASTRITIS.
3. Average duration about one year, rarely over two years.	3. Duration indefinite; may be for several years.	3. Duration indefinite.
4. Gastric hæmorrhage frequent, but rarely profuse; most common in the cachectic stage.	4. Gastric hæmorrhage less frequent than in cancer, but oftener profuse; not uncommon when the general health is but little impaired.	4. Gastric hæmorrhage rare.
5. Vomiting often has the peculiarities of that of dilatation of the stomach.	5. Vomiting rarely referable to dilatation of the stomach, and then only in a late stage of the disease.	5. Vomiting may or may not be present.
6. Free hydrochloric acid usually absent from the gastric contents in cancerous dilatation of the stomach.	6. Free hydrochloric acid usually present in the gastric contents.	6. Free hydrochloric acid may be present or absent.
7. Cancerous fragments may be found in the washings from the stomach or in the vomit (rare).	7. Absent.	7. Absent.
8. Secondary cancers may be recognized in the liver, the peritonæum, the lymphatic glands, and rarely in other parts of the body.	8. Absent.	8. Absent.
9. Loss of flesh and strength and development of cachexia usually more marked and more rapid than in ulcer or in gastritis, and less explicable by the gastric symptoms.	9. Cachectic appearance usually less marked and of later occurrence than in cancer, and more manifestly dependent upon the gastric disorders.	9. When uncomplicated, usually no appearance of cachexia.
10. Epigastric pain is often more continuous, less dependent upon taking food, less re	10. Pain is often more paroxysmal, more influenced by taking food, oftener relieved	10. The pain or distress induced by taking food is usually less severe than in cancer or

GASTRIC CANCER.	GASTRIC ULCER.	CHRONIC CATARRHAL GASTRITIS.
lieved by vomiting, and less localized than in ulcer.	by vomiting, and more sharply localized than in cancer.	ulcer. Fixed point of tenderness usually absent.
11. Causation not known.	11. Causation not known.	11. Often referable to some known cause, such as abuse of alcohol, gormandizing, and certain diseases, as phthisis, Bright's disease, cirrhosis of the liver, etc.
12. No improvement, or only temporary improvement, in the course of the disease.	12. Sometimes a history of one or more previous similar attacks. The course may be irregular and intermittent. Usually marked improvement by regulation of diet.	12. May be a history of previous similar attacks. More amenable to regulation of diet than is cancer.

Treatment.—The disease is incurable and palliative measures are alone indicated. The diet should consist of readily digested substances of all sorts. Many patients do best on milk alone. Washing out of the stomach, which may be done with a soft tube without any risk, is particularly advantageous when there is obstruction at the pylorus, and is by far the most satisfactory means of combatting the vomiting. The excessive fermentation is also best treated by lavage. When the pain becomes severe, particularly if it disturbs the rest at night, morphia must be given. One eighth of a grain, combined with carbonate of soda (gr. v), bismuth (gr. v–x), usually gives prompt relief, and the dose does not always require to be increased. Creosote (♏ j–ij) and carbolic acid are very useful. The bleeding in gastric cancer is rarely amenable to treatment. Operative measures have been advised and practised, and in exceptional instances there are cases in which the limited cancer could be resected with reasonable hope of recovery.

Non-cancerous tumors of the stomach rarely cause inconvenience. *Polypi* are common and they may be numerous; as many as one hundred and fifty have been reported in one case. *Sarcomata* are very rare. *Fibromata* and *lipomata* have been described.

Foreign bodies occasionally produce remarkable tumors of the stomach. The most extraordinary is the hair tumor, of which a number of instances have been reported in hysterical women who have been in the habit of eating their own hair. A specimen in the medical museum of

McGill University is in two sections, which form an exact mould of the stomach. The tumors which they form are large and very puzzling and have been mistaken for cancer. In one instance the ball of hair was removed by a surgical operation. The tumor was thought to be a movable kidney.

VIII. HÆMORRHAGE FROM THE STOMACH (*Hæmatemesis*).

Etiology.—Gastrorrhagia, as this symptom is called, may result from many conditions, some of which are local, others general.

1. In local disease in the stomach itself: (*a*) Cancer; (*b*) ulcer; (*c*) disease of the blood-vessels, such as miliary aneurisms of the smaller arteries, and occasionally varicose veins; (*d*) acute congestion, as in gastritis, and possibly in vicarious hæmorrhage, but both of these are extremely rare causes.

2. Passive congestion due to obstruction in the portal system. This may be either (*a*) hepatic, as in cirrhosis of the liver, thrombosis of the portal vein, or pressure upon the portal vein by tumor, and secondarily in cases of chronic disease of the heart and lungs; (*b*) splenic. Gastrorrhagia is by no means an uncommon symptom in enlarged spleen, and is explained by the intimate relations which exist between the vasa brevia and the splenic circulation.

3. Toxic: (*a*) The poisons of the specific fevers, small-pox, measles, yellow fever; (*b*) poisons of unknown origin, as in acute yellow atrophy and in purpura; (*c*) phosphorus.

4. Traumatism: (*a*) Mechanical injuries, such as blows and wounds, and occasionally by the stomach-tube; (*b*) the result of severe corrosive poisons.

5. Certain constitutional diseases: (*a*) Hæmophilia; (*b*) profound anæmias, whether idiopathic or due to splenic enlargements or to malaria; (*c*) cholæmia.

6. In certain nervous affections, particularly hysteria, and occasionally in progressive paralysis of the insane and epilepsy.

7. The blood may not come from the stomach, but flow into it. Thus it may pass from the nose or the pharynx. In hæmoptysis some of the blood may find its way into the stomach. The bleeding may take place from the œsophagus and trickle into the stomach, from which it is ejected. This occurs in the case of rupture of aneurism and of the œsophageal varices. A child may draw blood with the milk from the mother's breast even in considerable quantities and then vomit it.

8. Miscellaneous causes: Aneurism of the aorta or of its branches may rupture into the stomach. There are instances in which a patient has a single attack of hæmorrhage without even having a recurrence or without symptoms pointing to disease of the stomach.

In new-born infants hæmorrhage may occur within the first two weeks and prove rapidly fatal; the precise etiology of this is not known. This *melæna neonatorum*, according to Hecker, occurs in one of every five hundred infants. In a few instances it seems to be associated with an acquired or hereditary hæmophilia. Occasionally it is met with in sound, healthy infants; in others the birth has been premature, and in such cases the bleeding may be associated with premature interruption of the fœtal circulation. In very exceptional cases ulcer of the stomach has been found.

In medical practice, hæmorrhage from the stomach occurs most frequently in connection with cirrhosis of the liver and ulcer of the stomach. It is more frequent in women than in men, owing to the greater prevalence of round ulcer in the former.

Morbid Anatomy.—When death has occurred from the hæmatemesis there are signs of intense anæmia. The condition of the stomach varies extremely. The lesion is evident in cancer and in ulcer of the stomach. It is to be borne in mind that fatal hæmorrhage may come from a small miliary aneurism communicating with the surface by a pinhole perforation, or the bleeding may be due to the rupture of a submucous vein and the erosion in the mucosa may be small and readily overlooked. It may require a careful and prolonged search to avoid overlooking such lesions. In the large group associated with portal obstruction, whether due to hepatic or splenic disease, the mucosa is usually pale, smooth, and shows no trace of any lesion. In cirrhosis, fatal by hæmorrhage, one may sometimes search in vain for any focal lesion to account for the gastrorrhagia, and we must conclude that it is possible for even the most profuse bleeding to occur by diapedesis. The stomach may be distended with blood and the source of the hæmorrhage not apparent either in the stomach or in the the portal system. In such cases the œsophagus should be examined, as the bleeding may come from that source. In toxic cases there are invariably hæmorrhages in the mucous membrane itself.

Symptoms.—In rare instances fatal syncope may occur without any vomiting. In a case of the kind, in which the woman had fallen over and died in a few minutes, the stomach contained between three and four pounds of blood. The sudden profuse bleedings rapidly lead to profound anæmia. When due to ulcer or cirrhosis the bleeding usually recurs for several days. Fatal hæmorrhage from the stomach is met with in ulcer, cirrhosis, enlargement of the spleen, and in instances in which an aneurism ruptures into the stomach or œsophagus. Gastrorrhagia may occur in splenic anæmia or in leukæmia before the condition has aroused the attention of friends or physician.

The amount of blood lost is very variable, and in the course of a day the patient may bring up three or four pounds, or even more. In a case under the care of George Ross, in the Montreal General Hospital, the

patient lost during seven days ten pounds, by measurement, of blood. The usual symptoms of anæmia develop rapidly, and there may be slight fever, and subsequently œdema may occur. An interesting circumstance connected with gastro-intestinal hæmorrhage is the development of amaurosis, the mode of production of which is still under discussion.

Diagnosis.—In a majority of instances there is no question as to the origin of the blood. Occasionally it is difficult, particularly if the case has not been seen during the attack. Examination of the vomit readily determines whether blood is present or not. The materials vomited may be stained by wine, the juice of strawberries, raspberries, or cranberries, which give a color very closely resembling fresh blood, while iron and bismuth and bile may produce a blackish color like altered blood. In such cases the microscope will show clearly the presence of the shadowy outlines of the red blood-corpuscles, and, if necessary, spectroscopic and chemical tests may be applied.

Deception is sometimes practised by hysterical patients, who swallow and then vomit blood or colored liquids. With a little care such cases can usually be detected. The cases must be excluded in which the blood passes from the nose or pharynx, or in which infants swallow it with the milk.

There is not often difficulty in distinguishing between hæmoptysis and hæmatemesis, though the coughing and the vomiting are not infrequently combined. The following are points to be borne in mind in the diagnosis:

HÆMATEMESIS.	HÆMOPTYSIS.
1. Previous history points to gastric, hepatic, or splenic disease.	1. Cough or signs of some pulmonary or cardiac disease precedes, in many cases, the hæmorrhage.
2. The blood is brought up by vomiting, prior to which the patient may experience a feeling of giddiness or faintness.	2. The blood is coughed up, and is usually preceded by a sensation of tickling in the throat. If vomiting occurs, it follows the coughing.
3. The blood is usually clotted, mixed with particles of food, and has an acid reaction. It may be dark, grumous, and fluid.	3. The blood is frothy, bright red in color, alkaline in reaction. If clotted, rarely in such large coagula, and muco pus may be mixed with it.
4. Subsequent to the attack the patient passes tarry stools, and signs of disease of the abdominal viscera may be detected.	4. The cough persists, physical signs of local disease in the chest may usually be detected, and the sputa may be blood-stained for many days.

Prognosis.—Except in the case of rupture of aneurism or of large veins, hæmatemesis rarely proves fatal. In my experience death has fol-

lowed more frequently in cases of cirrhosis and splenic enlargement than in ulcer or cancer. In ulcer it is to be remembered that in the chronic hæmorrhagic form the bleeding may recur for years. The treatment of hæmatemesis is considered under gastric ulcer.

VII. DISEASES OF THE INTESTINES.

I. DISEASES OF THE INTESTINES ASSOCIATED WITH DIARRHŒA.

CATARRHAL ENTERITIS; DIARRHŒA.

In the classification of catarrhal enteritis the anatomical divisions of the bowel have been too closely followed, and a duodenitis, jejunitis, ileitis, typhlitis, colitis, and proctitis have been recognized; whereas in a majority of cases the entire intestinal tract, to a greater or lesser extent, is involved, sometimes the small most intensely, sometimes the large bowel, but during life it may be quite impossible to say which portion is specially affected.

Etiology.—The causes may be either *primary* or *secondary*. Among the causes of *primary* catarrhal enteritis are: (*a*) Improper food, one of the most frequent, especially in children, in whom it follows overeating, or the ingestion of unripe fruit. In some individuals special articles of diet will always produce a slight diarrhœa, which may not be due to a catarrh of the mucosa, but to increased peristalsis induced by the offending material. (*b*) Various toxic substances. Many of the organic poisons, such as those produced in the decomposition of milk and articles of food, excite the most intense intestinal catarrh. Certain inorganic substances, as arsenic and mercury, act in the same way. (*c*) Changes in the weather. A fall in the temperature of from twenty to thirty degrees, particularly in the spring or autumn, may induce—how, it is difficult to say—an acute diarrhœa. We speak of this as a catarrhal process, the result of cold or of chill. On the other hand, the diarrhœal diseases of children are associated in a very special way with the excessive heat of summer months. (*d*) Changes in the constitution of the intestinal secretions. We know too little about the *succus entericus* to be able to speak of influences induced by change in its quantity or quality. It has long been held that an increase in the amount of bile poured into the bowel might excite a diarrhœa; hence the term bilious diarrhœa, so frequently used by the older writers. Possibly there are conditions in which an excessive amount of bile is poured into the intestine, increasing the peristalsis, and hurrying on the contents; but the opposite state, a scanty secretion, by favoring the natural fermentative processes, much more commonly causes an intestinal catarrh. Absence of the pancreatic secretion from the intestine has been associated

in certain cases with a fatty diarrhœa. (*e*) Nervous influences. It is by no means clear how mental states act upon the bowels, and yet it is an old and trustworthy observation which every-day experience confirms that the mental state may profoundly affect the intestinal canal. These influences should not properly be considered under catarrhal processes, as they result simply from increased peristalsis or increased secretion, and are usually described under the heading *nervous diarrhœa*. In children it frequently follows fright. It is common, too, in adults as a result of emotional disturbances. Canstatt mentions a surgeon who always before an important operation had watery diarrhœa. In hysterical women it is seen as an occasional occurrence, due to transient excitement, or as a chronic, protracted diarrhœa, which may last for months or even years.

Among the *secondary* causes of intestinal catarrh may be mentioned: (*a*) Infectious diseases. Dysentery, cholera, typhoid fever, pyæmia, septicæmia, tuberculosis, and pneumonia are occasionally associated with intestinal catarrh. In dysentery and typhoid fever the ulceration is in part responsible for the catarrhal condition, but in cholera it is probably a direct influence of the bacilli or of the toxic materials produced by them. (*b*) The extension of inflammatory processes from adjacent parts. Thus, in peritonitis, catarrhal swelling and increased secretion are always present in the mucosa. In cases of invagination, hernia, tuberculous or cancerous ulceration, catarrhal processes are common. (*c*) Circulatory disturbances cause a catarrhal enteritis, usually of a very chronic character. This is common in diseases of the liver, such as cirrhosis, and in chronic affections of the heart and lungs—all conditions, in fact, which produce engorgement of the terminal branches of the portal vessels. (*d*) In the cachectic conditions met with in cancer, profound anæmia, Addison's disease, and Bright's disease intestinal catarrh may develop, and may terminate life.

Morbid Anatomy.—Changes in the mucous membrane are not always visible, and in cases in which, during life, the symptoms of intestinal catarrh have been marked, neither redness, swelling, nor increased secretion—the three signs usually laid down as characteristic of catarrhal inflammation—may be present post mortem. It is rare to see the mucous membrane injected; more commonly it is pale and covered with mucus. In the upper part of the small intestine the tips of the valvulæ conniventes may be deeply injected. Even in extreme grades of portal obstruction intense hyperæmia is not often seen. The entire mucosa may be softened and infiltrated, the lining epithelium swollen, or even shed, and appearing as large flakes among the intestinal contents. This is, no doubt, a post-mortem change. The lymph follicles are almost always swollen, particularly in children. The Peyer's patches may be prominent and the solitary follicles in the large and small bowel may stand out with distinctness and present in the centres little erosions, the so-called follicular ulcers. This may be a striking feature in the intestine in all forms of catarrhal enteritis in children, quite irrespective of the intensity of the diarrhœa.

When the process is more chronic the mucosa is firmer, in some instances thickened, in others distinctly thinned, and the villi and follicles present a slaty pigmentation.

Symptoms.—Acute and chronic forms may be recognized. The important symptom of both is diarrhœa, which, in the majority of instances, is the sole indication of this condition. It is not to be supposed that diarrhœa is invariably caused by, or associated with, catarrhal enteritis, as it may be produced by nervous and other influences. It is probable that catarrh of the jejunum may exist without any diarrhœa; indeed, it is a very common circumstance to find post mortem a catarrhal state of the small bowel in persons who have not had diarrhœa during life. The stools vary extremely in character. The color depends upon the amount of bile with which they are mixed, and they may be of a dark or blackish brown, or of a light-yellow, or even of a grayish-white tint. The consistence is usually very thin and watery, but in some instances the stools are pultaceous like thin gruel. Portions of undigested food can often be seen (lienteric diarrhœa), and flakes of yellowish-brown mucus. Microscopically there are innumerable micro-organisms, epithelium and mucous cells, crystals of phosphate of lime, oxalate of lime, and occasionally cholesterin and Charcot's crystals.

Pain in the abdomen is usually present in the acute catarrhal enteritis, particularly when due to food. It is of a colicky character, and when the colon is involved there may be tenesmus. More or less tympanites exists, and there are gurgling noises or borborygmi, due to the rapid passage of fluid and gas from one part to another. In the very acute attacks there may be vomiting. Fever is not, as a rule, present, but there may be a slight elevation of one or two degrees. The appetite is lost, there is intense thirst, and the tongue is dry and coated. In very acute cases, when the quantity of fluid lost is great and the pain excessive, there may be collapse symptoms. The number of evacuations varies from four or five to twenty or more in the course of the day. The attack lasts for two or three days, or may be prolonged for a week or ten days.

Chronic catarrh of the bowels may follow the acute form, or may develop gradually as an independent affection or as a sequence of obstruction in the portal circulation. It is characterized by diarrhœa, with or without colic. The dejections vary; when the small bowel is chiefly involved the diarrhœa is of a lienteric character, and when the colon is affected the stools are thin and mixed with much mucus. A special form of mucous diarrhœa will be subsequently described. The general nutrition of the patient in these chronic cases is greatly disturbed; there may be much loss of flesh and great pallor. The patients are inclined to suffer from low spirits, or hypochondriasis may develop.

Diagnosis.—It is important, in the first place, to determine, if possible, whether the large or small bowel is chiefly affected. In catarrh of the small bowel the diarrhœa is less marked, the pains are of a colicky

character, borborygmi are not so frequent, the fæces usually contain portions of food, and are more yellowish-green or grayish-yellow and flocculent and do not contain much mucus. When the large intestine is at fault there may be no pain whatever, as in the catarrh of the large intestine associated with tuberculosis and Bright's disease. When present, the pains are most intense and, if the lower portion of the bowel is involved, there may be marked tenesmus. The stools have a uniform soupy consistence, grayish in color and granular throughout, with here and there flakes of mucus, or they may contain very large quantities of mucus.

There are no positive symptoms by which the diagnosis of duodenitis can be made. It is usually associated with acute gastritis and, if the process extends into the bile-duct, with jaundice. Neither jejunitis nor ileitis can be separated from general intestinal catarrh.

ENTERITIS IN CHILDREN.

We may recognize three forms: (1) The acute dyspeptic diarrhœa; (2) cholera infantum; and (3) acute entero-colitis.

General Etiology of the Diarrhœas of Children.—The disease is most frequent in artificially fed children, and the greatest number of cases occur between the ages of six and eighteen months. A popular and well-founded belief ascribes special danger to the second summer of the infant. Infantile diarrhœa is very prevalent among the poorer classes in the large cities. It attacks, however, children with the most favorable surroundings. Two factors influence the disease, diet and temperature. An immense majority of all fatal cases are artificially fed. Of 1,943 fatal cases in Holt's statistics, only three per cent were exclusively breast fed. Among the poor the bowel complaint in children begins with the artificial feeding. The relation of temperature to the prevalence of diarrhœal diseases in children has long been recognized. The mortality curve begins to rise in May, increases in June, reaches the maximum in July, and gradually sinks through August and September. The maximum corresponds closely with the highest mean temperature; yet we cannot regard the heat itself as the direct agent, but only one of several factors. Thus the mean temperature of June is only four or five degrees lower than that of July, and yet the mortality is not more than one third. Seibert, who has carefully analyzed the mortality and the temperature, month by month, in New York, for ten years, fails to find a constant relation between the degree of heat and the number of cases of diarrhœa. Neither barometric pressure nor humidity appears to have any influence.

Relation of Bacteria.—The healthy fæces of sucklings contain a number of bacteria and micrococci, the most important of which are the *bacterium lactis aerogenes* and the *bacterium coli commune*. The former is only present in the intestine after a milk diet, the milk sugar appearing to furnish the materials necessary for its growth. It occurs more

in the upper portion of the bowel, and in this region excites the fermentative processes in the milk. The *bacterium coli commune* is found more abundantly in the lower portion of the small intestine and in the colon, and excites fermentative changes which are probably associated with certain phases of digestion. The observations of Escherich show the remarkable simplicity of this bacterial vegetation in the healthy fæces of milk-fed children, as these two alone develop and are constant. In infantile diarrhœa the number of bacteria which may be isolated from the stools is remarkable. Booker has discriminated forty varieties, the greatest number of which were found in the cases of cholera infantum. The two constant forms noted above do not disappear in the diarrhœal stools. No forms have been found to bear a constant or specific relation to the diarrhœal fæces, such as the two above mentioned do to the healthy milk fæces. The bacteria of the *proteus* group are most frequent, and possess pathogenic properties. All the varieties develop and produce important changes in the milk, which have been dealt with very fully by Booker in his studies. This author concludes that in the diarrhœa of infants "not one specific kind, but many different kinds of bacteria are concerned, and that their action is manifested more in the alteration of the food and intestinal contents and in the production of injurious products than in a direct irritation upon the intestinal wall." With these agree the conclusions of Jeffries and Baginsky regarding cholera infantum.

Morbid Anatomy.—We find most frequently a catarrhal swelling of the mucosa of both small and large bowel with enlargement of the lymph follicles. In more chronic cases the latter show small erosions or follicular ulcers; more rarely there is croupous enteritis affecting the lower part of the ileum and the colon. The changes in the other organs are neither numerous nor characteristic. Broncho-pneumonia occurs in many cases. The spleen may be swollen. Brain lesions are rare; the membranes and substance are often anæmic, but meningitis or thrombosis is very uncommon.

Clinical Forms.—Acute Dyspeptic Diarrhœa.—The child may appear in its usual health, but has an increase in the number of stools, without fever or special disturbance except slight restlessness at night. After persisting for a day or two the stools become more frequent and contain undigested food and curds, and are very offensive. In other cases the disease sets in abruptly with vomiting, griping pains, and fever, which may rise rapidly and reach 104° or 105°. There may be convulsions at the outset. The abdomen is sensitive, and the child lies with the legs drawn up. The stools consist of grayish or greenish-yellow fæces mixed with gas, curds, and portions of food. In children over two years of age such attacks not infrequently follow eating freely of unripe fruit or the drinking of milk which has been tainted. With judicious treatment the children improve in a few days; but relapses are not uncommon, and in the hot weather the attack may be the starting point of a severe entero-colitis. In a de-

bilitated child a mild attack may prove fatal. This dyspeptic diarrhœa is distinguished sharply from cholera infantum by the character of the stools, which never have a watery, serous character. In many instances this form precedes the onset of the specific fevers, particularly during the hot weather.

Cholera Infantum.—This is the counterpart in the infant of the so-called choleraic diarrhœa in the adult, and in their clinical aspects these two forms are identical. It is by no means so common as the ordinary dyspeptic diarrhœa of children, and, according to Holt, occurs only in two or three per cent of the cases of summer diarrhœa. It prevails in the hot weather and in children artificially fed or who have had previously some slight dyspeptic derangement. It is characterized by vomiting, uncontrollable diarrhœa, and collapse. The disease sets in with vomiting, which is incessant and is excited by any attempt to take food or drink. The stools are profuse and frequent; at first fæcal in character, brown or yellow in color, and finally thin, serous, and watery. The stools first passed are very offensive; subsequently they are odorless. The thin, serous stools are alkaline. There is fever, but the axillary temperature may register three or more degrees below that of the rectum. From the outset there is marked prostration; the eyes are sunken, the features pinched, the fontanelle depressed, and the skin has a peculiar ashy pallor. At first restless and excited, the child subsequently becomes heavy, dull, and listless. The tongue is coated at the onset, but subsequently becomes red and dry. As in all choleraic conditions, the thirst is insatiable; the pulse is rapid and feeble, and toward the end becomes irregular and imperceptible. Death may occur within twenty-four hours, with symptoms of collapse and great elevation of the internal temperature. Before the end the diarrhœa and vomiting may cease. In other instances the intense symptoms subside, but the child remains torpid and semi-comatose with fingers clutched, and there may be convulsions. The head may be retracted and the respirations interrupted, irregular, and of the Cheyne-Stokes type. The child may remain in this condition for some days without any signs of improvement. It was to this group of symptoms in infantile diarrhœa that Marshall-Hall gave the term "hydrencephaloid" or spurious hydrocephalus. As a rule, no changes in the brain or other organs are found, and the condition is no doubt caused by the toxic agents absorbed from the intestine. A remarkable condition of sclerema is described as a sequel of cholera infantum. The skin and subcutaneous tissues become hard and firm and the appearance has been compared to that of a half-frozen cadaver.

No constant organism has been found in these cases. Baginsky considers the disease the result of the action on the system of the poisonous products of decomposition encouraged by the various bacteria present—a *fäulniss* disease. The clinical picture is that produced by an acute bacterial infection, as in Asiatic cholera.

The *diagnosis* is readily made. There is no other intestinal affection in children for which it can be mistaken. The constant vomiting, the frequent watery discharges, the collapse symptoms, and the elevated temperature make an unmistakable clinical picture. The outlook in the majority of cases is bad, particularly in children artificially fed. Hyperpyrexia, extreme collapse, and incessant vomiting are the most serious symptoms.

Acute Entero-colitis.—In this form the ileum and colon are most affected, chiefly in the lymph follicles, hence the term follicular enteritis or follicular dysentery. It occurs most frequently in warm weather, in artificially fed children; but it may set in at any season of the year, and is the form of enteritis most common as a secondary complication in the specific fevers of childhood.

The attack may follow the ordinary dyspeptic diarrhœa. The temperature increases, the stools change in character and contain traces of blood and mucus, the former usually only in streaks. The fæces are passed without any pain. The abdomen is distended and tender along the line of the colon. Vomiting may be present at the outset, but is not a characteristic feature, as in cholera infantum. The diarrhœa may be gradually checked and convalescence is established in two or three weeks; in other instances the disease becomes subacute, the fever subsides, but the diarrhœa persists and the general health of the child rapidly deteriorates. The case may drag on for five or six weeks, when improvement gradually occurs or the child is carried off by a severe intercurrent attack. In a third form of acute entero-colitis, in which anatomically the lesions are those already mentioned—namely, an intense follicular inflammation—the symptoms are of a more severe character, and the affection is sometimes spoken of as acute dysentery. It attacks children up to the third or fourth year or even older. The onset is sudden, with high fever, vomiting, frequent stools, which at first contain remnants of food and fæces and subsequently much mucus and some blood. There is incessant pain, which may be more severe than in any intestinal affection of childhood. The prostration is very great and the fatal termination may occur within forty-eight hours. More commonly the case lasts for a week or longer. In two cases of this sort, in one of which death occurred in forty-eight and in the other in sixty-four hours, the anatomical characters were those of the most acute follicular enteritis, characterized by great swelling of the lymph follicles, some of which already presented necrotic foci.

The Cœliac Affection.—Under this heading Gee has described an intestinal disorder, most commonly met with in children between the ages of one and five, characterized by the occurrence of pale, loose stools, not unlike gruel or oatmeal porridge. They are bulky, not watery, yeasty, frothy, and extremely offensive. The affection has received various names, such as *diarrhœa alba* or *diarrhœa chylosa*. It is not associated with tuberculosis or other hereditary disease. It begins insidiously and there

are progressive wasting, weakness, and pallor. The belly becomes doughy and inelastic. There is often flatulency. Fever is usually absent. The disease is lingering and a fatal termination is common. So far nothing is known of the pathology of the disease. Ulceration of the intestines has been met with, but it is not constant. This affection resembles somewhat the disease in adults known as the *hill diarrhœa*, or the white flux of India; but certain of these tropical diarrhœas are, as will be mentioned, associated with the presence of the *anchylostoma*.

DIPHTHERITIC OR CROUPOUS ENTERITIS.

There are many conditions in which an intense croupous or diphtheritic inflammation of the mucosa of the small and large intestines occurs. It is met with most frequently, (*a*) as a secondary process in the infectious diseases—pneumonia, pyæmia in its various forms, and typhoid fever; (*b*) as a terminal process in many chronic affections, such as Bright's disease, cirrhosis of the liver, or cancer; and (*c*) as an effect of certain poisons—mercury, lead, and arsenic.

The disease presents three different anatomical pictures. In one group of cases the mucosa presents on the top of the folds a thin grayish-yellow diphtheritic exudate situated upon a deeply congested base. In some cases all grades may be seen between the thinnest film of superficial necrosis and involvement of the entire thickness of the mucosa. In the colon similar transversely arranged areas of necrosis are seen situated upon hypæramic patches, and it may be here much more extensive and involve a large portion of the membrane. There may be most extensive inflammation without any involvement of the solitary follicles of the large or small bowel.

In a second group of cases the membrane has rather a croupous character. It is grayish white in color, more flake-like and extensive, limited, perhaps, to the cæcum or to a portion of the colon; thus, in several cases of pneumonia I found this flaky adherent false membrane, in one instance forming patches 1 to 2 cm. in diameter, which were not unlike in form to rupia crusts.

In a third group the affection is really a follicular enteritis, involving the solitary glands, which are swollen and capped with an area of diphtheritic necrosis or are in a state of suppuration. Follicular ulcers are common in this form. The disease may run its course without any symptoms, and the condition is unexpectedly met with post mortem. In other instances there are diarrhœa, pain, but not often tenesmus or the passage of blood-stained mucus. In the toxic cases the intestinal symptoms may be very marked, but in the terminal colitis of the fevers and of constitutional affections the symptoms are often trifling.

PHLEGMONOUS ENTERITIS.

As an independent affection this is excessively rare, even less frequent than its counterpart in the stomach. It is seen occasionally in connection with intussusception, strangulated hernia, and chronic obstruction. Apart from these conditions it occurs most frequently in the duodenum, and leads to suppuration in the submucosa and abscess formation. Except when associated with hernia or intussusception the affection cannot be diagnosed. The symptoms usually resemble those of peritonitis.

MUCOUS COLITIS.

This affection is known by various names, such as *membranous enteritis*, *tubular diarrhœa*, and *mucous colic*. It is a remarkable disease, to which much attention has been paid for several centuries. An exhaustive description of it is given by Woodward, in Vol. II of the Medical and Surgical Reports of the Civil War. It is an affection of the large bowel, characterized by the production of a very tenacious adherent mucus, which may be passed in long strings or as a continuous, tubular membrane. I have twice had opportunities of seeing this membrane *in situ*, closely adherent to the mucosa of the colon, but capable of separation without any lesion of the surface. Judging from the statement of English authors as to its rarity, it would appear to be a more frequent disease in this country. According to W. A. Edwards, 80 per cent of the recorded adult cases have been in women. It occurs occasionally in children. Of 111 cases six were under the age of ten. The cases are almost invariably seen in nervous or hysterical women or in men with neurasthenia. All grades of the affection occur, from the passage of a slimy mucus, like frog-spawn, to large tubular casts a foot or more in length. Microscopically the casts are, as shown by Sir Andrew Clark, not fibrinous, but mucoid, and even the firmest consist of dense, opaque, transformed mucus. It is due to a derangement of the mucous glands of the colon, the nature of which is quite unknown.

Symptoms.—The disease persists for years, varying extremely from time to time, and is characterized by paroxysms of pain in the abdomen, tenderness, occasionally tenesmus, and the passage of flakes or long strings of mucus, sometimes of definite casts of the bowel. The attacks last for a day or, in some instances, for ten days or two weeks. Mental emotions and worry of any sort seem particularly apt to bring on an attack. Occasionally errors in diet or dyspepsia precede an outbreak. Membranes are not passed with every paroxysm, even when the pains and cramps are severe. There are instances in which the morphia habit has been contracted on account of the severity of the pain. There may be marked nervous symptoms, and authors mention hysterical outbreaks, hypochondriasis, and melancholia.

The *diagnosis* is rarely doubtful, but it is important not to mistake the membranes for other substances; thus, the external cuticle of asparagus and undigested portions of meat or sausage-skins sometimes assume forms not unlike mucous casts, but the microscopical examination will quickly differentiate them.

ULCERATIVE ENTERITIS.

In addition to the specific ulcers of tuberculosis, syphilis, and typhoid fever, the following forms of ulceration occur in the bowels:

(*a*) *Follicular Ulceration.*—As previously mentioned, this is met with very commonly in the diarrhœal diseases of children, and also in the secondary or terminal inflammations in many fevers and constitutional disorders. The ulcers are small, punched out, with sharply cut edges, and they are usually limited to the follicles. With this form may be placed the catarrhal ulcers of some writers.

(*b*) *Stercoral Ulcers*, which occur in long-standing cases of constipation. Very remarkable indeed are the cases in which the sacculi of the colon become filled with rounded small scybala, some of which produce distinct ulcers in the mucous membrane. The fæcal masses may have lime salts deposited in them, and thus form little enteroliths.

(*c*) *Simple Ulcerative Colitis.*—This affection, which clinically is characterized by diarrhœa, is often regarded wrongly as a form of dysentery. It is not a very uncommon affection, and is most frequently met with in men above the middle period of life. The ulceration may be very extensive, so that a large proportion of the mucosa is removed. The lumen of the colon is sometimes greatly increased, and the muscular walls hypertrophied. There are instances in which the bowel is contracted. Frequently the remnants of the mucosa are very dark, even black, and there may be polypoid outgrowths between the ulcers.

These cases rarely come under observation at the outset, and it is difficult to speak of the mode of origin. They are characterized by diarrhœa of a lienteric rather than of a dysenteric character. There is never blood or pus in the stools. Constipation may alternate with the diarrhœa. There is usually great impairment of nutrition, and the patients get weak and sallow. Perforation occasionally occurs.

The disease may prove fatal, or it may pass on and become chronic. The affection was not very infrequent at the Philadelphia Hospital, and though the disease bears some resemblance to dysentery, it is to be separated from it. Some of the cases which we have learned to recognize as amœbic dysentery resemble this form very closely. An excellent description of it is given by Hale White.*

(*d*) *Ulceration from External Perforation.*—This may result from the

* Guy's Hospital Reports, 1888.

erosion of new growths or, more commonly, from localized peritonitis with abscess formation and perforation of the bowel. This is met with most frequently in tuberculous peritonitis, but it may occur in the abscess which follows perforation of the appendix or suppurative or gangrenous pancreatitis. Fatal hæmorrhage may result from the perforation.

(*e*) *Cancerous Ulcers.*—In very rare instances of multiple cancer or sarcoma the submucous nodules break down and ulcerate. In one case the ileum contained eight or ten sarcomatous ulcers secondary to an extensive sarcoma in the neighborhood of the shoulder-joint.

(*f*) Occasionally a *solitary ulcer* is met with in the cæcum or colon, which may lead to perforation. Two instances of ulcer of the cæcum, both with perforation, have come under my observation, and in one instance a simple ulcer of the colon perforated and led to fatal peritonitis.

Diagnosis of Intestinal Ulcers.—As a rule, diarrhœa is present in all cases, but exceptionally there may be extensive ulceration, particularly in the small bowel, without diarrhœa. Very limited ulceration in the colon may be associated with frequent stools. The character of the dejections is of great importance. Pus, shreds of tissue, and blood are the most valuable indications. Pus occurs most frequently in connection with ulcers in the large intestine, but when the bowel alone is involved the amount is rarely great, and the passage of any quantity of pure pus is an indication that it has come from without, most commonly from the rupture of a pericæcal abscess, or in women an abscess of the broad ligament. Pus may also be present in cancer of the bowel, or it may be due to local disease in the rectum. A purulent mucus may be present in the stools in cases of ulcer, but it has not the same diagnostic value. The swollen, sago-like masses of mucus which are believed by some to indicate follicular ulceration are met with also in mucous colitis. Hæmorrhage is an important and valuable symptom of ulcer of the bowel, particularly if profuse. It occurs under so many conditions that taken alone it may not be specially significant, but with other coexisting circumstances it may be the most important indication of all.

Fragments of tissue are occasionally found in the stools in ulcer, particularly in the extensive and rapid sloughing in dysenteric processes. Definite portions of mucosa, shreds of connective tissue, and even bits of the muscular coat may be found. Pain occurs in many cases, either of a diffuse, colicky character, or sometimes, in the ulcer of the colon, very limited and well defined.

Perforation is an accident liable to happen when the ulcer extends deeply. In the small bowel it leads to a localized or general peritonitis. In the large intestine, too, a fatal peritonitis may result, or if perforation takes place in the posterior wall of the ascending or descending colon, the production of a large abscess cavity in the retro-peritonæum. In a case at the University Hospital, Philadelphia, there was a perforation at

the splenic flexure of the colon with an abscess containing air and pus —a condition of subphrenic pyo-pneumothorax.

Treatment of the Previous Conditions.

(*a*) **Acute Dyspeptic Diarrhœa.**—All solid food should be withheld. If vomiting is present ice may be given, and small quantities of milk and soda water may be taken. If the attack has followed the eating of large quantities of undigestible material, castor oil or calomel is advisable, but is not necessary if the patient has been freely purged. If the pain is severe, twenty drops of laudanum and a drachm of spirits of chloroform may be given, or, if the colic is very intense, a hypodermic of a quarter of a grain of morphia. It is not well to check the diarrhœa unless it is profuse, as it usually stops spontaneously within forty-eight hours. If persistent, the aromatic chalk powder or large doses of bismuth (thirty to forty grains) may be given. A small enema of starch (two ounces) with twenty drops of laudanum, every six hours, is a most valuable remedy.

(*b*) **Chronic Diarrhœa,** including chronic catarrh and ulcerative enteritis. It is important, in the first place, to ascertain, if possible, the cause and whether ulceration is present or not. So much in treatment depends upon the careful examination of the stools—as to the amount of mucus, the presence of pus, the occurrence of parasites, and, above all, the state of digestion of the food—that the practitioner should pay special attention to them. Many cases simply require rest in bed and a restricted diet. Chronic diarrhœa of many months' or even of several years' duration may be sometimes cured by strict confinement to bed and a diet of boiled milk and albumen water.

In that form in which immediately after eating there is a tendency to loose evacuations it is usually found that some one article of diet is at fault. The patient should rest for an hour or more after meals. Sometimes this alone is sufficient to prevent the occurrence of the diarrhœa. In those forms which depend upon abnormal conditions in the small intestine, either too rapid peristalsis or faulty fermentative processes, bismuth is indicated. It must be given in large doses—from half a drachm to a drachm three times a day. The smaller doses are of little use. Naphthalin preparations here do much good, given in doses of from ten to fifteen grains (in capsule) four or five times a day. Larger doses may be needed. Salol and the salicylate of bismuth may be tried.

An extremely obstinate and intractable form is the diarrhœa of hysterical women. A systematic rest cure will be found most advantageous, and if a milk diet is not well borne the patient may be fed exclusively on egg albumen. The condition seems to be associated in some cases with increased peristalsis, and in such the bromides may do good, or preparations of opium may be necessary. There are instances which prove most obstinate and resist all forms of treatment, and the patient may be greatly reduced. A change of air and surroundings may do more than medicines.

In a large group of the chronic diarrhœas the mischief is seated in the

colon and is due to ulceration. Medicines by the mouth are here of little value. The stools should be carefully watched and a diet arranged which shall leave the smallest possible residue. Boiled or peptonized milk may be given, but the stools should be examined to see whether there is an excess of food or of curds. Meat is, as a rule, badly borne in these cases. The diarrhœa is best treated by enemata. The starch and laudanum should be tried, but when ulceration is present it is better to use astringent injections. From two to four pints of warm water containing from half a drachm to a drachm of nitrate of silver may be used. In the chronic diarrhœa which follows dysentery this is particularly advantageous. In giving large injections the patient should be in the dorsal position, with the hips elevated, and it is best to allow the injection to flow in gradually from a siphon bag. In this way the entire colon can be irrigated and the patient can retain the injection for some time. The silver injections may be very painful, but they are invaluable in all forms of ulcerative colitis. Acetate of lead, boracic acid, sulphate of copper, sulphate of zinc, and salicylic acid may be [illegible] in one per cent solutions.

In mucous colitis no benefit can be expected from remedies administered by the mouth. The topical applications should be made to the mucous membrane of the colon by the enemata just mentioned, and the general nervous condition should receive appropriate treatment.

In the intense forms of choleraic diarrhœa in adults associated with constant vomiting and frequent watery discharges the patient should be given at once a hypodermic of a quarter of a grain of morphia, which should be repeated in an hour if the pains return or the purging persists. This gives prompt relief, and is often the only medicine needed in the attack. The patient should be given stimulants, and, when the vomiting is allayed by suitable remedies, small quantities of milk and lime water.

(*c*) **The Diarrhœa of Children.**—*Hygienic management* is of the first importance. The effect of a change from the hot, stifling atmosphere of a town to the mountains or the sea is often seen at once in a reduction in the number of stools and a rapid improvement in the physical condition. Even in cities much may be done by sending the child into the parks or for daily excursions on the water. However extreme the condition, fresh air is indicated. The child should not be too thickly clad. Many mothers, even in the warm weather, clothe their children too heavily. Bathing is of value in infantile diarrhœa, and when the fever rises above 102·5° the child should be placed in a warm bath, the temperature of which may be gradually reduced, or the child is kept in the bath for twenty minutes, by which time the water is sufficiently cooled. Much relief is obtained by the application of ice-cold cloths or of the ice-cap to the head. Irrigation of the colon with ice-cold water is sometimes favorable, but it has not the advantage of the general bath, the beneficial effect of which is seen, not only in the reduction of the temperature, but in a general stimulation of the nervous system of the child.

Dietetic Treatment.—In the case of a hand-fed child it is important, if possible, to get a wet-nurse. While fever is present, digestion is sure to be much disturbed, and the amount of food should be restricted. If water or barley water be given the child will not feel the deprivation of food so much. When the vomiting is incessant it is much better not to attempt to give milk or other articles of food, but let the child take the water whenever it will.

In the dyspeptic diarrhœas of infants, practically the whole treatment is a matter of artificial feeding, and there is no subject in medicine on which it is more difficult to lay down satisfactory rules. No doubt within a few years the study of the bacterial processes going on in the intestines of the child will give us most important suggestions. From his observations Escherich lays down the following rules, recognizing two well-defined forms of intestinal fermentation—the acid and the alkaline: If there is much decomposition, with foul, offensive stools, the albuminous articles should be withheld from the diet and the carbohydrates given, such as dextrin foods, sugar, and milk, which, on account of its sugar, ranks with the carbohydrates. If there is acid fermentation, with sour but not fetid stools, an albuminous diet is given, such as broths and egg albumen. It is, however, by no means certain whether the reaction of the stools, upon which this author relies, is a sufficient test of the nature of the intestinal fermentation. In the dyspeptic diarrhœas of artificially fed infants it is best, as a rule, to withhold milk and to feed the child, for the time at least, on egg albumen, broths, and beef juices. To prepare the egg albumen, the whites of two or three eggs may be stirred in a pint of water and a teaspoonful of brandy and a little salt mixed with it. The child will usually take this freely, and it is both stimulating and nourishing. It is sometimes remarkable with what rapidity a child which has been fed on artificial food and milk will pick up and improve on this diet alone. Beef-juice is obtained by pressing with a lemon-squeezer fresh steak, previously minced and either uncooked or slightly broiled. This may be given alternately with the egg albumen or it may be given alone. Mutton or chicken broth will be found equally serviceable, but it is prepared with greater difficulty and contains more fat. In the preparation, a pound of mutton, chicken, or beef, carefully freed from fat, is minced and placed in a pint of cold water and allowed to stand in a glass jar on ice for three or four hours. It should then be cooked over a slow fire for at least three hours, then strained, allowed to cool, the fat skimmed off, sufficient salt added, and it may then be given either warm or cold. These naturally prepared albumen foods are very much to be preferred to the various artificial substances. There is no form of nourishment so readily assimilated and apt to cause so little disturbance as egg albumen or the simple beef juices. The child should be fed every two hours, and in the intervals water may be freely given. It cannot be expected that, with the digestion seriously impaired, as much food can be taken as in health,

and in many instances we see the diarrhœa aggravated by persistent overfeeding. When the child's stomach is quieted and the diarrhœa checked there may be a gradual return to the milk diet. The milk should be sterilized, and in institutions and in cities this simple prophylactic measure is of the very first importance and is readily carried out by means of the Arnold steam sterilizer. The milk should be at first freely diluted—four parts of water to one of milk, which is perhaps the preferable way—or it may be peptonized. The stools should be examined daily, as important indications may be obtained from them. Milk-whey and forms of fermented milk are sometimes useful and may be employed when the stomach is very irritable. These general directions as to food also hold good in cholera infantum.

Medicinal Treatment.—The first indication in the dyspeptic diarrhœa of children is to get rid of the decomposing matter in the stomach and intestines. The diarrhœa and vomiting partially effect this, but it may be more thoroughly accomplished, so far as the stomach is concerned, by irrigation. It may seem a harsh procedure in the case of young infants, but in reality, with a large-sized soft-rubber catheter, it is practised without any difficulty. By means of a funnel, lukewarm water is allowed to pass in and out until it comes away quite clear. I can speak in the very warmest manner of the good results obtained by this simple procedure in cases of the most obstinate gastro-intestinal catarrh in children. In most cases the warm water is sufficient. In some hands this method has probably been carried to excess, but that does not detract from its great value in suitable cases. To remove the fermenting substances from the intestines, doses of calomel or gray powder may be administered. The castor oil is equally efficacious, but is more apt to be vomited. Irrigation of the large bowel is useful, and not only thoroughly removes fermenting substances, but cleanses the mucosa. The child should be placed on the back with the hips elevated. A flexible catheter is passed for from six to eight inches and from a pint to two pints of water allowed to flow in from a fountain syringe. A pint will thoroughly irrigate the colon of a child of six months and a quart that of a child of two years. The water may be lukewarm, but when there is high fever ice-cold water may be used. In cases of entero-colitis there may be injections with borax, a drachm to the pint, or dilute nitrate of silver, which may be either given in large injections, as in the adult, or in injections of three or four ounces with three grains of nitrate of silver to the ounce. These often cause very great pain, and it is well in such cases to follow the silver injection with irrigations of salt solution, a drachm to a pint.

We are still without a reliable intestinal antiseptic. Neither naphthalin, salol, resorcin, the salicylates, nor mercury meets the indications. As in the diarrhœa of adults, bismuth in large doses is often very effective, but practitioners are in the habit of giving it in doses which are quite insufficient. To be of any service it must be used in large doses, so that an

infant a year old will take as much as two drachms in the day. The gray powder has long been a favorite in this condition and may be given in half-grain doses every hour. It is perhaps preferable to calomel, which may be used in small doses of from one tenth to one fourth of a grain every hour at the onset of the trouble. The sodium salicylate (in doses of two or three grains every two hours to a child a year old) has been recommended.

In cholera infantum serious symptoms may develop with great rapidity, and here the incessant vomiting and the frequent purging render the administration of remedies extremely difficult. Irrigation of the stomach and large bowel is of great service, and when the fever is high ice-water injections may be used or a graduated bath. As in the acute choleraic diarrhœa of adults, morphia hypodermically is the remedy which gives greatest relief, and in the conditions of extreme vomiting and purging, with restlessness and collapse symptoms, this drug alone commands the situation. A child of one year may be given from $\frac{1}{100}$ to $\frac{1}{80}$ of a grain, to be repeated in an hour, and again if not better. When the vomiting is allayed, attempts may be made to give gray powder in half-grain doses with $\frac{1}{10}$ of Dover's powder. Starch (℥ ij) and laudanum (♏ij–iij) injections, if retained, are soothing and beneficial. The combination of bismuth with Dover's powder will also be found beneficial. No attempt should be made to give food. Water may be allowed freely, even when ejected at once by vomiting. Small doses of brandy or champagne, frequently repeated and given cold, are sometimes retained. When the collapse is extreme, hypodermic injections of one per cent saline solution may be used as recommended in Asiatic cholera, and hypodermic injections of ether and brandy may be tried. The convalescence requires very careful management, as many cases pass on into the condition of entero-colitis. When the intense symptoms have subsided, the food should be gradually given, beginning with teaspoonful doses of egg albumen or beef-juice. It is best to withhold milk for several days, and when used it should be at first completely peptonized or diluted with gruel. A teaspoonful of raw, scraped meat three or four times a day is often well borne.

II. MISCELLANEOUS AFFECTIONS OF THE BOWELS.

Dilatation of the Colon.—This may be general or localized to the sigmoid flexure.

It occurs not infrequently as a transient condition, and in many cases it has an important influence, inasmuch as the distention may be extreme, pushing up the diaphragm and seriously impairing the action of the heart and lungs. H. Fenwick has called attention to this as occasionally a cause of sudden heart-failure.

Dilatation of the sigmoid flexure occurs particularly when this portion of the bowel is congenitally very long. In such cases the bowel may be so distended that it occupies the greater part of the abdomen, pushing up the liver and the diaphragm. An acute condition is sometimes caused by a twist in the mesocolon.

There is a chronic form in which the gut reaches an enormous size. The coats may be hypertrophied without evidence of any special organic change in the mucosa. In a specimen which I saw with W. E. Hughes, in Philadelphia, the colon was enormously dilated and held fourteen pints of water, and the sigmoid flexure was four inches in diameter. It was removed from a boy, aged three, who had had obstinate constipation and at the age of two an attack of entero-colitis. At one time he was nineteen days without a passage; on another occasion twenty-four. The abdomen was enormously distended, everywhere tympanitic. The hypertrophy of the bowel-wall was much greater toward the sigmoid flexure than near the cæcum. In the section on Constipation in Infants a case is referred to in which the colon and sigmoid flexure appeared to be dilated.

Infarction of the Bowel.—The mesenteric vessels are terminal arteries, and when blocked by emboli or thrombi the condition of infarction follows in the territory supplied. Probably the occlusion of small vessels does not produce any symptoms and the circulation may be re-established. If the superior mesenteric artery is blocked a serious and fatal condition follows. Three instances have come under my observation. In one, a woman aged fifty-five was seized with nausea and vomiting, which persisted for more than a week. There was pain in the abdomen, tympanites, and toward the close the vomiting was incessant and fæcal. The autopsy showed great congestion, with swelling and infiltration of the jejunum and ileum. The superior mesenteric artery was blocked at its orifice by a firm thrombus. In the second case, a woman aged seventy-five was seized with severe abdominal pain and frequent vomiting. At first there was diarrhœa; subsequently the symptoms pointed to obstruction, with great distention of the abdomen. The post-mortem showed the small bowel, with the exception of the first foot of the jejunum and the last six inches of the ileum, greatly distended and deeply infiltrated with blood. The mesentery was also congested and infiltrated. The superior mesenteric artery contained a firm brownish-yellow clot. There were many recent warty vegetations on the mitral valve. In the third case, a man aged forty was suddenly seized with intense pain in the abdomen, became faint, fell to the ground, and vomited. For a week he had persistent vomiting, severe diarrhœa, tympanites, and great pain in the abdomen. The stools were thin and at times blood-tinged. The autopsy showed an aneurism involving the aorta at the diaphragm. The superior mesenteric artery, half an inch from its origin on the sac, was blocked by a portion of the fibrinous clot of the aneurism. In the horse, infarction of the intestine is

extremely common in connection with the verminous aneurisms of the mesenteric arteries and is the usual cause of colic in this animal.

III. APPENDICITIS.

(*Typhlitis and Perityphlitis*).

This is one of the most important of intestinal affections. Unfortunately, much confusion still exists about the forms of inflammation in the cæcal region. Thus there are recognized *typhlitis*, inflammation of the cæcum itself; *perityphlitis*, inflammation of the peritonæum covering the cæcum; *paratyphlitis*, inflammation of the connective tissue behind the cæcum, or, more correctly, as the cæcum is usually covered by a serous membrane, of the connective tissue in the neighborhood of this part of the bowel. The use of the last two terms should be altogether discarded, as the cases are, with rare exceptions, due to disease of the appendix vermiformis, and not to affections of the cæcum.

We have in the cæcal region the following affections:

Typhlitis, inflammation of the cæcum proper—a doubtful and uncertain malady, the pathology of which is unknown, but which clinically is still recognized by authorities. A majority of the cases are unquestionably due to appendix disease.

Appendicitis: (1) Catarrhal; (2) ulcerative; (3) perforative, with the production of abscesses, which may be pericæcal, pelvic, intra-peritoneal, perinephritic, or lumbar, depending on the situation of the vermiform process.

TYPHLITIS.

At present inflammation of any sort, accompanied by pain in the right iliac fossa, is generally thought to be due to disease of the appendix; and, so far as post-mortem statistics indicate, an immense majority of all these cases are due to this cause. Clinically, however, authors still recognize typhlitis (inflammation of the cæcum), associated with lodgment of fæces (*typhlitis stercoralis*) The cases are met with in young persons, in boys more commonly than in girls; the subjects have usually been constipated, or there have been errors in diet. The patient complains of pain in the right iliac fossa; there are constipation, nausea, sometimes vomiting; fever, if present, is usually slight, rarely rising above 101°. There is fulness in the right iliac fossa, the decubitus is dorsal, and the right thigh may be flexed. On pressure there is tenderness, and in many instances a doughy, sausage-shaped tumor in the right flank. The attack lasts for from three days to a week, the pain gradually subsides, the tumor mass disappears, and recovery is complete.

The anatomical condition is unknown, and it is by no means certain that these cases are in reality cæcal. Many are probably due to dis-

ease of the appendix, and even when the sausage-shaped, doughy tumor, regarded as diagnostic of typhlitis stercoralis, is present, the cæcitis and fæcal retention may be secondary. The cases do well; a great majority of them terminate favorably, a point which, as Pepper remarks, is opposed to the belief that they are all dependent upon appendix disease.

In the *treatment* of this condition an ice-bag should be placed over the cæcal region, large enemata given once or twice a day to empty the colon, and opium given to allay the pain.

More serious disease of the cæcum does occasionally occur, and there are a few instances in which an ulcer perforates. The rarity of this, however, is shown by the fact that Fitz was only able to collect three cases. Two instances have come under my observation in which perforation of an ulcer in the cæcum led to extensive pericæcal abscess.

APPENDICITIS.

The appendix vermiformis is extremely variable in position. It commonly lies behind the ileum with the tip pointing toward the spleen. It is frequently turned up behind the cæcum or it lies upon the psoas muscle with its tip at the margin of the pelvis. It has, however, been found in almost every region of the abdomen. Thus in my post-mortem notes it is stated to have been found in close contact with the bladder; adherent to the ovary or broad ligament; in the central portion of the abdomen, close to the navel; in contact with the gall-bladder; passing out at right angles and adherent to the sigmoid flexure to the left of the middle line of the abdomen; and in one case it passed with the cæcum into the inguinal canal, curved upon itself, re-entered the abdomen, and was adherent to the wall of an abscess cavity just to the right of the promontory of the sacrum. Foreign bodies rarely lodge in it. Only two instances have come under my notice; in one there were eight snipe shot and in the other five apple pips. On the other hand, oval bodies resembling date stones are very common. They consist of inspissated mucus and fæces, in which in time lime salts are deposited, forming enteroliths.

Post-mortem examinations show that the appendix is very frequently the seat of extensive disease, past or present, without the history of any definite symptoms pointing to trouble in the cæcal region. Among the commonest of these conditions is obliteration, either total or partial. When at the cæcal end, the appendix may be enormously dilated, forming a tumor the size of the thumb or as large as a sausage. In the cases of obliteration the appendix may be free, more commonly it is adherent, and there may be about it signs of old inflammation or even a small encapsulated abscess, which has given no trouble.

Etiology.—Appendicitis is a disease of young persons. According to Fitz's statistics, more than fifty per cent of the cases occur before the twentieth year; sixty per cent between the sixteenth and thirtieth years

(Einhorn). It has been met with as early as the seventh week, but it is rarely seen prior to the third year. It is very much more common in males than in females—eighty per cent, according to the tables of Fitz, but in his personal experience in 72 cases males were only twice as frequently affected as females. Contrary to the general experience, the Munich figures (Einhorn) indicate a relatively greater number of women attacked. The fæcal concretions and foreign bodies already referred to probably play the most important *rôle* in the etiology of the disease. In a series of 152 cases the fæcal masses were present in forty-seven per cent and foreign bodies in twelve per cent. Matterstock, in 169 cases of perforative appendicitis, found the percentage to be fifty-three and twelve, respectively. Typhoid fever and tuberculosis frequently induce ulceration of the appendix, but not often perforation. Fitz suggests that some of the cases of peritonitis which recover in typhoid fever are due to perforation of the appendix. Traumatism plays a very definite *rôle*, and in a number of cases the symptoms have followed the lifting of a heavy weight, or a fall or a blow. Constipation, overloading the stomach with indigestible food, indiscretions in diet, are mentioned in many cases. The tendency of the disease to recur is remarkable. Among 257 cases (Fitz) eleven per cent had had previous attacks. In the recurring appendicitis no factor is of greater importance than overeating, and attacks may follow directly upon the taking of large quantities of unsuitable food.

Morbid Anatomy.—For practical purposes we recognize a catarrhal and an ulcerative appendicitis. In *catarrhal appendicitis* the entire tube is thickened, the peritoneal surface may be slightly injected, and adhesions may have formed, so that there is a slight circumscribed peritonitis. The lumen may be much contracted, particularly toward the cæcal end; the mucosa is thickened, covered with a tenacious mucus; and very commonly fæcal concretions or small enteroliths are present. The coats are thickened throughout, particularly the muscularis, and the entire tube is firm and stiff. It may attain the size of the index finger or even that of the thumb. When laid open longitudinally, it at once assumes a rolled form in the reverse direction.

Ulceration and Perforation of Appendix.—Many cases of ulcer present no symptoms. In typhoid fever and phthisis eleven instances have come under my observation in which there were no clinical indications of the lesion. The dangerous ulcers follow the irritation of the fæcal concretions or foreign bodies. It may result also from obliteration of the cæcal end and distention of the lumen with fluid. The perforation may have the following direct effects: (*a*) The appendix may hang free in the peritoneal cavity, adhesions not having formed, when the perforation at once excites a diffuse and violent suppurative peritonitis.

(*b*) More commonly, in fact, almost as a rule, the ulcerated appendix becomes adherent and a localized peritonitis results. Perforation then occurs, with the formation of a circumscribed intraperitoneal abscess

cavity, which may be small and which varies in situation with the appendix. Perhaps the most common situation is on the psoas muscle, in the neighborhood of the terminal portion of the ileum. In cases of this sort I have most frequently found the small localized abscess just at the angle between the ileum and the cæcum. It may, however, be within the pelvis or close to the sacrum. Adhesive peritonitis, perforation, and the formation of a localized abscess may go on without the production of serious symptoms, and the condition may be found when death has resulted from accident or some intercurrent affection. In some cases a large circumscribed fæcal abscess forms in the iliac region and points midway between the navel and the anterior superior spine of the ilium.

Unfortunately, in many cases the localized abscess cavity excites the most intense peritonitis. Often without actual rupture diffuse suppurative disease occurs. In many instances the first indication of serious trouble is the acute, agonizing pain which follows the diffusion of this localized peritoneal process. The contents of the limited abscess may not be more than a few cubic centimetres, are usually darkish gray in color, and excessively offensive.

(*c*) When the appendix passes behind the cæcum and colon and is not within the peritonæum, perforation at once produces a retroperitoneal abscess, which may terminate in many different ways; thus the pus may pass beneath the iliacus fascia and appear at Poupart's ligament, in which situation external perforation may occur and recovery take place. The pus may be chiefly in the retroperitoneal tissue in the flank, forming a large perinephritic abscess. In a case under the care of Gardner, of Montreal, an enormous abscess cavity developed in this situation, which contained air, pushed up the diaphragm nearly to the second rib, and produced the symptoms of pneumothorax. Perforation of the pleura may occur in these cases, forming a fæcal pleural fistula. The pus may extend along the psoas muscle and may perforate the hip joint, or pass to the neighborhood of the rectum, or produce multiple abscesses of the scrotum, or, passing through the obturator foramen, form a large gluteal abscess. Perforation into the bladder may occur, but is not nearly so common as perforation into the bowel. In both instances recovery may follow, though there is greater danger in perforation into the latter. The appendix has been discharged *per anum*.

The remote effects of perforative appendicitis are interesting. Hæmorrhage may occur. In one of my cases the appendix was adherent to the promontory of the sacrum, and the abscess cavity had perforated in two places into the ileum. Death resulted from profuse hæmorrhage. Cases are on record in which the internal iliac artery or the deep circumflex iliac artery has been opened. Suppurative pylephlebitis may result from inflammation of the mesenteric veins near the perforated appendix. Two instances of it have come under my notice; in one there was a small localized abscess which had resulted from the perforation of a typhoid ulcer

of the appendix. In the other case, which I saw with Machell, of Toronto, the symptoms were those of septicæmia and suppuration of the liver. The abscess of the appendix was small and had not produced symptoms. In the healing of extensive inflammation about the margin of the pelvis the iliac veins may be greatly compressed, and one of my patients had for months œdema of the right leg, which is still enlarged.

Symptoms.—As already mentioned, a simple catarrhal appendicitis may lead to a fatal result, and, on the other hand, perforation and abscess formation may take place without exciting serious symptoms. No classification into light, medium, and severe forms can be made, as the most severe of all features of the disease—general peritonitis—may be the very first indication of the existence of any trouble.

Catarrhal inflammation may induce the most characteristic features of appendix disease. The facts on which this statement is made are conclusive. A man aged twenty-eight was admitted to the Johns Hopkins Hospital with pains in the abdomen, localized in the right iliac fossa, which in July became severe enough to confine him to bed for several weeks. In August the attack returned with severity. No tumor was to be felt externally, but on rectal examination a firm, rounded body could be felt high up on the right margin of the pelvis. Laparotomy was performed and the appendix found in the true pelvis, slightly adherent, very much thickened, but without perforation or ulceration. Bridge reports an instance in which a woman aged twenty-eight had an attack of severe abdominal pain, vomiting, constipation, but no tumor. The temperature rose as high as 101°, the thighs were flexed, and there was pain on extension of the psoas. Temporary improvement followed and then a recurrence, accompanied with rise of temperature and return of the pain. Laparotomy was performed and a thickened, dense appendix found, which contained three small enteroliths. In both these instances persistent, severe symptoms were caused by what must be termed a chronic inflammation of the appendix, without ulceration and without perforation. Both cases recovered. A similar instance has occurred at the Pennsylvania Hospital, under the care of Thomas G. Morton. A suppurative peritonitis may also occur without perforation or ulceration. In a case reported by Fitz there had been previous attacks, from which recovery by resolution had taken place; then an abscess at the brim of the pelvis was opened and drained. After recovery again a recurrence occurred, and finally the appendix was removed and found to be thickened, but neither ulcerated nor perforated, and only adherent in a limited extent to the omentum.

In *perforative appendicitis* there may be initial symptoms, such as nausea, constipation, sometimes diarrhœa, and a sense of uneasiness and distress in the right iliac fossa. These may possibly be associated with the localized peritonitis. A sudden violent pain in the abdomen, most commonly in the right iliac fossa, is the "most constant, first decided symp-

tom of perforating inflammation of the appendix," and occurred in eighty-four per cent of the cases analyzed by Fitz. It is usually limited to the fossa, but sometimes extends toward the navel or to the perinæum, testicle, or thigh. Fever, furred tongue, and vomiting may precede or accompany this pain. An initial chill is rare. The temperature ranges from 101° to 103°; sometimes it is higher; the pulse is increased in frequency. The patient in walking bends over, favors the right side, and has difficulty in standing straight. When in bed the patient usually lies with the right leg drawn up and complains of pain on extension. Micturition may be frequent or there may be retention of urine. Diarrhœa seems to be more frequent in children than in adults.

Physical Signs.—Tympanites may be early and interfere considerably with the examination. On the other hand, the abdomen may be flat, hard, and board-like even with diffuse peritonitis. In a great majority of the cases there is tenderness in the right iliac fossa and over the region of the appendix. McBurney has called attention to the value of a special localized point of tenderness on deep pressure situated from one and a half to two inches from the anterior superior spine of the ileum on a line drawn between this point and the navel. When firm, continuous pressure is made with one finger at this point the pain may be of the most exquisite character. Circumscribed swelling may be present, but it is inconstant and is not found in more than one half the cases. It is usually in the fossa below a line passing from the anterior superior spine to the navel and two or three finger-breadths above Poupart's ligament. In many instances it is a diffuse thickening and induration; in others a well-defined tumor mass can be detected. If there is much tension of the abdominal muscles and pain, it is best to make a thorough examination under ether. In the cases in which the abscess is large, fluctuation may be felt above Poupart's ligament or in the flank, and in some instances crepitation. Dulness is not present unless the exudation is abundant and superficial. Usually the small localized tumors are entirely masked by the distended intestines. A rectal examination should be made in every instance. When the appendix is above the brim of the pelvis it cannot be reached, but when, as so often happens, it curls over into the pelvis, it or the thickened indurated area about it may be felt. After all, the great danger is not so much in the limited peritonitis which results from the perforation, as in the extension of it to the general peritonæum. In Fitz's analysis, the second, third, and fourth days included the largest number of cases of beginning peritonitis. General abdominal pain, tympanites and an aggravation of the general symptoms indicate the onset of this serious complication.

Diagnosis.—Appendicitis is by far the most common inflammatory condition producing symptoms, not only in the cæcal region but in the abdomen, generally in persons under thirty. Laparotomy has taught us that, almost without exception, sudden pain in the right iliac fossa with

fever, localized tenderness with or without tumor, means appendix disease. Almost the only other local condition to be differentiated is stercoral cæcitis, which is characterized by less severe pain, slighter fever, and the presence of an elongated doughy mass in the lumbar region; it must be remembered that in many of these cases the appendix is probably affected.

Perinephritic and pericæcal abscess from perforation of ulcer, either simple or cancerous, and circumscribed peritonitis in this region from other causes can rarely be differentiated until an exploratory incision is made.

Catarrhal and perforative appendicitis cannot always be differentiated, as the cases which I have quoted show that in intensity of pain, severity of symptoms, and even in the production of peritonitis, the two may be identical.

Briefly stated, localized pain in the right iliac fossa with or without induration or tumor, the existence of McBurney's tender point, fever, furred tongue, vomiting, constipation or diarrhœa, indicate appendicitis. The occurrence of general peritonitis is suggested by increase and diffusion of the abdominal pain, tympanites (as a rule), marked aggravation of the constitutional symptoms, particularly elevation of fever and increased rapidity of the pulse. Alonzo Clark's sign, obliteration of hepatic dulness, is rarely present, as the peritonæum in these cases does not often contain gas.

The hypodermic needle should never be used unless there is marked tumor with dulness on percussion in the cæcal region.

Intussusception and internal strangulation may present very similar symptoms, and if the patient is only seen at the latter stages, when there is diffuse peritonitis and great tympany, the features may be almost identical. Fæcal vomiting, which is common in obstruction, is never seen in appendicitis, and in children the marked tenesmus and bloody stools are important signs of intussusception. It is not often difficult when the cases are seen early and when the history is clear, but mistakes have been made by surgeons of the first rank.

In women, disease of the tubes and pelvic peritonitis from any cause may simulate appendicitis; but the history and the local examination, under ether, should in most cases enable the practitioner to discriminate between these conditions. In neurotic patients the odd and anomalous symptoms produced by floating kidney may be thought to be due to appendicitis.

Prognosis.—If we regard every case of inflammation in the cæcal region as appendicitis, a large proportion of the cases recover. The gravity of the disease is difficult to estimate, but it certainly must be ranked as one of the most serious and fatal of the abdominal affections of young persons. Post-mortem observations show that very many instances get well, often without treatment. As mentioned, recurrence is common, so much so that over forty per cent of the cases may be spoken of as recurrent ap-

pendicitis. Sixty-eight per cent of the fatal cases die during the first eight days. Extension to the general peritonæum is almost always fatal. Perforation into the bowel is often followed by recovery. Perforation externally is still less serious. Nowadays, with the prompt surgical interference, the prognosis is very much better.

Treatment.—The studies of Pepper, Noyes, With, and Matterstock, and more particularly the elaborate and thorough study of Fitz, have directed the attention of physicians to the clinical features of the diseases in the cæcal region, but to the surgeons we owe invaluable lessons relating to diagnosis and, above all, to treatment.

The suggestion of Willard Parker with reference to early operation has been carried out and advocated by Sands, Bull, and Weir in New York, by Morton and Keen in Philadelphia, and by Treves in London.

Treatment of the Attack.—The medical treatment of appendicitis can be expressed in three words—rest, opium, and enemata. The patient should be quiet in bed with an ice-bag placed in the right iliac fossa. If there is much pain, opium should be given either hypodermically or by the mouth. Medium-sized injections of warm water may be given twice daily. I would protest most earnestly against the indiscriminate use of saline purges, which have been advocated under a total misapprehension. It cannot be too strongly emphasized that, as a rule, the initial condition, which produces the pain, the fever, and the local signs, is the establishment after perforation of a localized peritonitis. So long as the abscess cavity remains limited, resolution is possible. Saline purges mean more or less disturbance of the local conditions and a definite increase in the risk of general peritonitis. It is an entirely different matter when this is established. Salines in some instances then do good, but in appendicitis, when the general peritonæum is involved, the mischief is done, and neither salines nor laparotomy materially influence the result.

The profession has yet to learn the lesson that perforative appendicitis is in more than three fourths of all cases a surgical affection, and perhaps the most important function of the physician, under whose care the disease always comes at first, is to say whether the case is suitable and when the operation should be performed.

Operation is indicated: (*a*) in all cases of acute inflammatory trouble in the cæcal region when, whether tumor is present or not, the general symptoms are severe, as shown by tympany, spreading pain, increase in fever, and increase in the rapidity of the pulse. In so many of the cases no tumor is to be felt that stress cannot be laid upon its absence.

(*b*) When a definite tumor is present, associated with attacks such as have been described, particularly if they have been recurrent. An occasional exception may be made to this rule when, even with small tumor, the symptoms rapidly subside and the patient improves. We are here on the horns of a dilemma. On the one hand, it is in just such cases that perforation and fatal peritonitis may at any moment occur, and, on the

other, the tumor may gradually disappear and the patient may have no further trouble.

(*c*) In recurrent appendicitis, when the attacks are of such severity and frequency as seriously to interrupt the patient's occupation. Is the interim operation advisable or shall the patient be advised to wait until an attack? Opinions differ on this point. It is best, I think, to wait. The operation has risks; patients have died from the interim laparotomy; and there is always a chance that the recovery from an attack may prove permanent. Both clinical observation and morbid anatomy show that complete healing is by no means rare. The physician must be guided too by the character of the surgical techinque at his command, and could hand over his patient without qualms to a modern operator whose success has demonstrated the safety of his methods.

IV. INTESTINAL OBSTRUCTION.

Intestinal obstruction may be caused by strangulation, intussusception, twists and knots, strictures and tumors, and by abnormal contents.

Etiology and Pathology.—(*a*) **Strangulation.**—This is the most frequent cause of acute obstruction, and occurred in thirty-four per cent of the 295 cases analyzed by Fitz,* and in thirty-five per cent of the 1,134 cases of Leichtenstern.† Of the 101 cases of strangulation in Fitz's table, which has the special value of having been carefully selected from the literature since 1880, the following were the causes: Adhesions, 63; vitelline remains, 21; adherent appendix, 6; mesenteric and omental slits, 6; peritoneal pouches and openings, 3; adherent tube, 1; pedunculer tumor, 1. The bands and adhesions result, in a majority of cases, from former peritonitis. A number of instances have been reported following operations upon the pelvic organs in women. The strangulation may be recent and due to adhesion of the bowel to the abdominal wound or a coil may be caught between the pedicle of a tumor and the pelvic wall. Such cases are only too common. Late occlusion after recovery from the operation is due to bands and adhesions.

The vitelline remains are represented by Meckel's diverticulum, which forms a finger-like projection from the ileum, usually within eighteen inches of the ileo-cæcal valve. It is a remnant of the omphalo-mesenteric duct, through which, in the early embryo, the intestine communicated with the yolk-sac. The end, though commonly free, may be attached to the abdominal wall near the navel, or to the mesentery, and a ring is thus formed through which the gut may pass.

Seventy per cent of the cases of obstruction from strangulation occur

* Transactions of the Congress of American Physicians and Surgeons, vol. i, 1889. The percentages of his tables are used throughout this section.

† Von Ziemssen's Encyclopædia of Practical Medicine.

in males; forty per cent of all the cases occur between the ages of fifteen and thirty years. In ninety per cent of the cases of obstruction from these causes the site of the trouble is in the small bowel; the position of the strangulated portion was in the right iliac fossa in sixty-seven per cent of the cases, and in the lower abdomen in eighty-three per cent.

(*b*) **Intussusception.**—In this condition one portion of the intestine slips into an adjacent portion, forming an invagination or intussusception. The two portions make a cylindrical tumor, which varies in length from a half-inch to a foot or more. The condition is always a descending intussusception, and as the process proceeds, the middle and inner layers increase at the expense of the outer layer. An intussusception consists of three layers of bowel: the outermost, known as the intussuscipiens, or receiving layer; a middle or returning layer; and the innermost or entering layer. The student can obtain a clear idea of the arrangement by making the end of a glove-finger pass into the lower portion. The actual condition can be very clearly studied in the post-mortem invaginations which are so common in the small bowel of children. In the statistics of Fitz, 93 of 295 cases of acute intestinal obstruction were due to this cause. Of these, 52 were in males and 27 in females. The cases are most common in early life, thirty-four per cent under one year and fifty-six per cent under the tenth year. No definite causes could be assigned in 42 of the cases; in the others diarrhœa or habitual constipation had existed.

The site of the invagination varies. We may recognize (1) an *ileo-cæcal*, when the ileo-cæcal valve descends into the colon. There are cases in which this is so extensive that the valve has been felt per rectum. This form occurred in seventy-five per cent of the cases. In the *ileo-colic* the lower part of the ileum passes through the ileo-cæcal valve. (2) The *ileal*, in which the ileum is alone involved. (3) The *colic*, in which it is confined to the large intestine. And (4) *colico-rectal*, in which the colon and rectum are involved.

Irregular peristalsis is the essential cause of intussusception. Nothnagel found in the localized peristalsis caused by the faradic current that it was not the descent of one portion into the other, but the drawing up of the receiving layer by contraction of the longitudinal coat. Invagination may follow any limited, sudden, and severe peristalsis.

In the post-mortem examination, in a case of death from intussusception, the condition is very characteristic. Peritonitis may be present or an acute injection of the serous membrane. When death occurs early, as it may do from shock, there is little to be seen. The portion of bowel affected is large and thick, and forms an elongated tumor with a curved outline. The parts are swollen and congested, owing to the constriction of the mesentery between the layers. The entire mass may be of a deep livid-red color. If very recent there is only congestion, and perhaps a slight layer of lymph, and the intussusception can be reduced, but when it

has lasted for a few days, lymph is thrown out, the layers are glued together, and the entering portion of the gut cannot be withdrawn.

The anatomical condition accounts for the presence of the tumor, which exists in two thirds of all cases; and the engorgement, which results from the compression of the mesenteric vessels, explains the frequent occurrence of blood in the discharges, which has so important a diagnostic value. If the patient survives, necrosis and sloughing of the invaginated portion may occur, and if union has taken place between the middle and outer layer, the calibre of the gut may be restored and a cure in this way effected. Many cases of the kind are on record. In the Museum of the Medical Faculty of McGill University are 17 inches of small intestine, which were passed by a lad who had had symptoms of internal strangulation, and who made a complete recovery.

(*c*) **Twists and Knots.**—Volvulus or twist occurred in 42 of the 295 cases. Sixty-eight per cent were in males. It is most frequent between the ages of thirty and forty. In the great majority of all cases the twist is axial and associated with an unusually long mesentery. In fifty per cent of the cases it was in the sigmoid flexure. The next most common situation is about the cæcum, which may be twisted upon its axis or bent upon itself. As a rule, in volvulus the loop of bowel is simply twisted upon its long axis, and the portions at the end of the loop cross each other and so cause the strangulation. It occasionally happens that one portion of the bowel is twisted about another.

(*d*) **Strictures and Tumors.**—These are very much less important causes of acute obstruction, as may be judged by the fact that there are only 15 instances out of the 295 cases, in 14 of which the obstruction occurred in the large intestine. On the other hand, they are common causes of chronic obstruction.

The obstruction may result from: (1) *Congenital stricture.* These are exceedingly rare. Much more commonly the condition is that of complete occlusion, either forming the imperforate anus or the congenital defect by which the duodenum is not united to the pylorus. (2) *Simple cicatricial stenosis*, which results from ulceration, tuberculous or syphilitic, more rarely from dysentery, and most rarely of all from typhoid ulceration. (3) *New growths.* The malignant strictures are due chiefly to cylindrical epithelioma, which forms an annular tumor, most commonly met with in the large bowel, about the sigmoid flexure, or the descending colon. Of benign growths, papillomata, adenomata, lipomata, and fibromata occasionally induce obstruction. (4) *Compression and traction.* Tumors of neighboring organs, particularly of the pelvic viscera, may cause obstruction by adhesion and traction; more rarely, a coil, such as the sigmoid flexure, filled with fæces, compresses and obstructs a neighboring coil. In the healing of tuberculous peritonitis the contraction of the thick exudate may cause compression and narrowing of the coils.

(*e*) **Abnormal Contents.**—Foreign bodies, such as fruit stones, coins, pins, needles, or false teeth, are occasionally swallowed accidentally, or by lunatics on purpose. Round worms may become rolled into a tangled mass and cause obstruction. In reality, however, the majority of foreign bodies, such as coins, buttons, and pins, swallowed by children, cause no inconvenience whatever, but in a day or two are found in the stools. Occasionally such a foreign body as a pin will pass through the œsophagus and will be found lodged in some adjacent organ, as in the heart (Peabody), or a barley ear may reach the liver (Dock).

Medicines, such as magnesia or bismuth, have been known to accumulate in the bowels and produce obstruction, but in the great majority of the cases the condition is caused by fæces, gall-stones, or enteroliths. Of 44 cases, in 23 the obstruction was by gall-stones, in 19 by fæces, and in 2 by enteroliths. Obstruction by fæces may happen at any period of life. As mentioned when speaking of dilatation of the colon, it may occur in young children and persist for weeks. In fæcal accumulation the large bowel may reach an enormous size and the contents become very hard. The retained masses may be channeled, and small quantities of fæcal matter are passed until a mass too large enters the lumen and causes obstruction. There may be very few symptoms, as the condition may be borne for weeks or even for months.

Obstruction by gall-stones is not very infrequent, as may be gathered from the fact that twenty-three cases were reported in the literature in eight years. Eighteen of these were in women and five in men. In six sevenths of the cases it occurred after the fiftieth year. The obstruction is usually in the ileo-cæcal region, but it may be in the duodenum. These large solitary gall-stones ulcerate through the gall-bladder, usually into the small intestine, occasionally into the colon. In the latter case they rarely cause obstruction. Courvoisier has collected one hundred and thirty-one cases in the literature.

Enteroliths may be formed of masses of hair, more commonly of the phosphates of lime and magnesia, with a nucleus formed of a foreign body or of hardened fæces. Nearly every museum possesses specimens of this kind. They are not so common in men as in ruminants, and, as indicated in Fitz's statistics, are very rare causes of obstruction.

Symptoms.—(*a*) **Acute Obstruction.**—Constipation, pain in the abdomen, and vomiting are the three important symptoms. Pain sets in early and may come on abruptly while the patient is walking or, more commonly, during the performance of some action. It is at first colicky in character, but subsequently it becomes continuous and very intense. Vomiting follows quickly and is a constant and most distressing symptom. At first the contents of the stomach are voided, and then greenish, bile-stained material, and soon, in cases of acute and permanent obstruction, the material vomited is a brownish-black liquid, with a distinctly fæcal odor. This sequence of gastric, bilious, and, finally, stercoraceous vomit-

ing is perhaps the most important diagnostic feature of acute obstruction. The constipation may be absolute, without the discharge of either fæces or gas. Very often the contents of the bowel below the stricture are discharged. Distention of the abdomen usually occurs, and when the large bowel is involved it is extreme. On the other hand, if the obstruction is high up in the small intestine, there may be very slight tympany. At first the abdomen is not painful, but subsequently it may become acutely tender.

The constitutional symptoms from the outset are severe. The face is pallid and anxious, and finally collapse symptoms supervene. The eyes become sunken, the features pinched, and the skin is covered with a cold, clammy sweat. The pulse becomes rapid and feeble. There may be no fever; the axillary temperature is often subnormal. The tongue is dry and parched and the thirst is incessant. The urine is high-colored, scanty, and there may be suppression, particularly when the obstruction is high up in the bowel. This is probably due to the constant vomiting and the small amount of liquid which is absorbed. The case terminates as a rule in from three to six days. In some instances the patient dies from shock or sinks into coma.

(*b*) **Symptoms of Chronic Obstruction.**—When due to fæcal impaction, there is a history of long-standing constipation. There may have been discharge of mucus, or in some instances the fæcal masses have been channeled, and so have allowed the contents of the upper portion of the bowel to pass through. In elderly persons this is not infrequent; but examination, either *per rectum* or externally, in the course of the colon, will reveal the presence of hard scybalous masses. There may be retention of fæces for weeks without exciting serious symptoms. In other instances there are vomiting, pain in the abdomen, gradual distention, and finally the ejecta become fæcal. The hardened masses may excite an intense colitis or even peritonitis.

In stricture, whether cicatricial or cancerous, the symptoms of obstruction are very diverse. Constipation gradually comes on, is extremely variable, and it may be months or even years before there is complete obstruction. There are transient attacks, in which from some cause the fæces accumulate above the stricture, the intestine becomes greatly distended, and in the swollen abdomen the coils can be seen in active peristalsis. In such attacks there may be vomiting, but it is very rarely of a fæcal character. In the majority of these cases the general health is seriously impaired; the patient gradually becomes anæmic and emaciated, and finally, in an attack in which the obstruction is complete, death occurs with all the features of acute occlusion or the case may be prolonged for ten or twelve days.

Diagnosis.—(*a*) **The Situation of the Obstruction.**—Hernia must be excluded, which is by no means always easy, as fatal obstruction may occur from the involvement of a very limited portion of the gut in the

external ring or in the obturator foramen. Mistakes from both of these causes have come under my observation; they were cases in which it was impossible to make a diagnosis other than acute obstruction. Timely operation would have saved both lives. A thorough rectal and vaginal examination should be made, which will give important information as to the condition of the pelvic and rectal contents, particularly in cases of intussusception, in which the descending bowel can sometimes be felt. In cases of obstruction high up the empty coils sink into the pelvis and can there be detected. Rectal exploration with the entire hand is of doubtful value. In the inspection of the abdomen there are important indications, as the special prominence in certain regions, the occurrence of definite, well-defined masses, and the presence of hypertrophied coils in active peristalsis. In obstruction in the duodenum or jejunum there may only be slight distention in the upper part of the abdomen, associated usually with rapid collapse and anuria.

In the ileum and cæcum the distention is more in the central portion of the abdomen; the vomiting is distinctly fæcal and occurs early. In obstruction of the colon, tympanites is much more extensive and general. Tenesmus is more common, with the passage of mucus and blood. The course is not so quick, the collapse does not supervene so rapidly, and the urinary secretion is not so much reduced.

In obstruction from stricture or tumor the situation can in some cases be accurately localized, but in others it is very difficult. Digital examination of the rectum should first be made. The rectal tube may then be passed, but it is impossible to get beyond the sigmoid flexure. In the use of the rigid tube there is danger of perforation of the bowel in the neighborhood of a stricture. The quantity of fluid which can be passed into the large intestine should be estimated. The capacity of the large bowel is about six quarts. The safe limits of pressure have been determined to be under ten feet in an infant and twenty feet in an adult. To thoroughly irrigate the bowel the patient should be chloroformed and should lie on the back or on the side; best on the back with the hips elevated. Treves suggests that the cæcal region should be auscultated during the passage of the fluid. For diagnostic purposes the rectum may be inflated, either by the bellows or by the use of bicarbonate of soda and tartaric acid. In certain cases these measures give important indications as to the situation of the obstruction in the large bowel.

(*b*) **Nature of the Obstruction.**—This is often difficult, not infrequently impossible, to determine. *Strangulation* is not common in very early life. In many instances there have been previous attacks of abdominal pain, or there are etiological factors which give a clew, such as old peritonitis or operation on the pelvic viscera. Neither the onset nor the character of the pain gives us any information. In rare instances nausea and vomiting may be absent. The vomiting usually becomes fæcal from the third to the fifth day. A tumor is not common in strangulation, and

was present in only one fifth of the cases. Fever is not of diagnostic value.

Intussusception is an affection of childhood, and is of all forms of internal obstruction the one most readily diagnosed. The presence of tumor, bloody stools, and tenesmus are the important factors. The tumor is usually sausage-shaped and felt in the region of the transverse colon. It existed in 66 of 93 cases. It was present on the first day in more than one third of the cases, on the second day in more than one fourth, and on the third day in more than one fifth. Blood in the stools occurs in at least three fifths of the cases, either spontaneously or following the use of an enema. The blood may be mixed with mucus. Tenesmus is present in one third of the cases. Fæcal vomiting is not very common and was present in only 12 of the 93 instances. Abdominal tympany is a symptom of slight importance, occurring in only one third of the cases.

Volvulus can rarely be diagnosed. The frequency with which it involves the sigmoid flexure is to be borne in mind. The passage of a flexible tube or injecting fluids might in these cases give valuable indications. An absolute diagnosis can probably be made only by an abdominal section.

In fæcal obstruction the condition is usually clear, as the fæces can be felt per rectum and also in the distended colon. Fæcal vomiting, tympany, abdominal pain, nausea, and vomiting are late and are not so constant. In obstruction by gall-stone a few of the cases gave a previous history of gall-stone colic. Jaundice was present in only two of the twenty-three cases. Pain and vomiting, as a rule, occur early and are severe, and fæcal vomiting is present in two thirds of the cases. A tumor is rarely evident.

(*c*) **Diagnosis from other Conditions.**—Acute enteritis with great relaxation of the intestinal coils, vomiting, and pain may be mistaken for obstruction. In an autopsy on a case of this kind the small and large bowels were intensely inflamed, relaxed, sodden, and enormously distended. The symptoms were those of acute obstruction, but the intestine was free from duodenum to rectum. Of late years many instances have been reported in which peritonitis following disease of the appendix has been mistaken for acute obstruction. The intense vomiting, the general tympany and abdominal tenderness, and in some instances the suddenness of the onset are very deceptive, and in two cases which have come under my notice the symptoms pointed very strongly to internal strangulation. In appendix disease the temperature is more frequently elevated, the vomiting is never fæcal, and in many cases there is a history of previous attacks in the cæcal region. Acute hæmorrhagic pancreatitis may produce symptoms which simulate closely intestinal obstruction. A boy was admitted to the Johns Hopkins Hospital with a history of obstinate vomiting, intense abdominal pain, gradually increasing tympany, and no passage for several days. His condition seemed serious and he was transferred at once to the surgical wards. At the operation the coils were found uniformly

distended and covered in places with the thinnest film of lymph. No obstruction existed, but there was a tumor-like mass surrounding the pancreas, firm, hard, and deeply infiltrated with blood. The patient improved after the operation and recovered completely.

Treatment.—Purgatives should not be given. For the pain hypodermics of morphia are indicated. To allay the distressing vomiting, the stomach should be washed out. Not only is this directly beneficial, but Küssmaul claims that the abdominal distention is relieved, the pressure in the bowel above the seat of obstruction is lessened, and the violent peristalsis is diminished. It may be practised three or four times a day, and in some instances has proved beneficial; in others curative. Thorough irrigation of the large bowel with injections should be practised, the fluid being allowed to flow in from a siphon syringe, and the amount carefully estimated. Jonathan Hutchinson recommends that the patient be placed under an anæsthetic, the abdomen thoroughly kneaded, and a copious enema given while in the inverted position. Then, with the aid of three or four strong men, the patient is to be thoroughly shaken, first with the abdomen held downward, and subsequently in the inverted position.

Inflation may also be tried, by forcing the air into the rectum with the bellows or with a Davidson's syringe. It is a measure not without risk, as instances of rupture of the bowel have been reported. Fitz's figures show that in the first eight years of the last decade there were thirty-three cases of recovery after injection or inflation in cases of certain or probable intussusception, and eleven deaths. In cases of acute obstruction, if these means do not prove successful by the third day, surgical measures should be resorted to, and when the obstruction seems persistent and the condition serious, laparotomy should be performed at once.

For the tympanites turpentine stupes and hot applications may be applied; if extreme, the bowel may be punctured with a small aspirator needle. In cases of chronic obstruction the diet must be carefully regulated, and opium and belladonna are useful for the paroxysmal pains. Enemata should be employed, and if the obstruction becomes complete, resort must be had to surgical measures.

V. CONSTIPATION (*Costiveness*).

Definition.—Retention of fæces from any cause.

Constipation in Adults.—The causes are varied and may be classed as general and local.

General Causes.—(*a*) Constitutional peculiarities: Torpidity of the bowels is often a family complaint and is found more often in dark than in fair persons. (*b*) Sedentary habits, particularly in persons who eat too much and neglect the calls of nature. (*c*) Certain diseases, such as anæmia, neurasthenia and hysteria, chronic affections of the liver, stomach,

and intestines, and the acute fevers. Under this heading may appropriately be placed that most injurious of all habits, *drug-taking*. (*d*) Either a coarse diet, which leaves too much residue, or a diet which leaves too little may be a cause of costiveness.

Local Causes.—Weakness of the abdominal muscles in obesity or from overdistention in repeated pregnancies. Atony of the large bowel from chronic disease of the mucosa; the presence of tumors, physiological or pathological, pressing upon the bowel; enteritis; foreign bodies, large masses of scybala, and strictures of all kinds. By far the most important local cause is atony of the colon, particularly of the muscles of the sigmoid flexure by which the fæces are propelled into the rectum.

Symptoms.—The most persistent constipation for weeks or even months may exist with fair health. All kinds of evils have been attributed to poisoning by the resorption of noxious matters from the retained fæces—copræmia—but it is not likely that this takes place to any extent. Chlorosis, which Sir Andrew Clark attributes to fæcal poisoning, is not always associated with constipation, and if due to this cause should be in men, women, and children the most common of all disorders. Debility, lassitude, and mental depression are frequent symptoms in constipation, particularly in persons of a nervous temperament. Headache, loss of appetite, and a furred tongue may also occur. Individuals differ extraordinarily in this matter; one feels wretched all day without the accustomed evacuation; another is comfortable all the week except on the day on which by purge or enema the bowels are relieved.

When persistent, the accumulation of fæces leads to unpleasant, sometimes serious symptoms, such as piles, ulceration of the colon, distention of the sacculi, perforation, enteritis, and occlusion. In women pressure may cause pain at the time of menstruation and a sensation of fulness and distention in the pelvic organs. Neuralgia of the sacral nerves may be caused by an overloaded sigmoid flexure. The fæces collect chiefly in the colon. Even in extreme grades of constipation it is rare to find dry fæces in the cæcum. The fæces may form large tumors at the hepatic or splenic flexures, or a sausage-like, doughy mass above the navel, or an irregular lumpy tumor in the left inguinal region. In old persons the sacculi of the colon become distended and the scybala may remain in them and undergo calcification, forming enteroliths.

In cases with prolonged retention the fæcal masses become channelled and diarrhœa may occur for days before the true condition is discovered by rectal or external examination. In women who have been habitually constipated, attacks of diarrhœa with nausea and vomiting should excite suspicion and lead to a thorough examination of the large bowel. Fever may occur in these cases, and Meigs has reported an instance in which the condition simulated typhoid fever.

Constipation in infants is a common and troublesome disorder. The causes are congenital, dietetic, and local. There are instances in which

the child is constipated from birth and may not have a natural movement for years and yet thrive and develop. An instance of the kind was in my ward recently in which a baby of seven months had never had a movement without preliminary injections. The abdomen became swollen every day, but subsided after an injection and the passage of a long catheter. No stricture could be felt. I have already referred to a case of W. E. Hughes's, in which there was enormous dilatation of the large bowel with persistent constipation. In some of these patients there may be constricting bands, or, as in a case of Cheever's, a congenital stricture.

Dietetic causes are more common. In sucklings it often arises from an unnatural dryness of the small residue which passes into the colon, and it may be very difficult to decide whether the fault is in the mother's milk or in the digestion of the child. Most probably it is the latter, as some babies may be persistently costive on natural or artificial foods. Too much casein in the milk is believed by some writers to be the cause. In older children it is of the greatest importance that regular habits should be enjoined. Carelessness on the part of the mother in this matter often lays the foundation of troublesome constipation in after life. Impairment of the contractibility of the intestinal wall in consequence of inflammation, disturbance in the normal intestinal secretions, and mechanical obstruction by tumors, twists, and intussusception are the chief local causes.

Treatment.—Much may be done by systematic habits, particularly in the young. The desire to go to stool should always be granted. Exercise in moderation is helpful. In stout persons and in women with pendulous abdomens the muscles should have the support of a bandage. Friction or regularly applied massage is invaluable in the more chronic cases. A good substitute is a metal ball weighing from four to six pounds, which may be rolled over the abdomen every morning for five or ten minutes. The diet should be light, with plenty of fruit and vegetables, particularly salads and tomatoes. Oatmeal is usually laxative, though not to all; brown bread is better than that made from fine white flour. Of liquids, water and the aërated mineral waters may be taken freely. A tumblerful of cold water on rising, taken slowly, is efficacious in many cases. A glass of hot water at night may also be tried alone. A pipe or a cigar after breakfast is with many men an infallible remedy.

When the condition is not very obstinate it is well to try to relieve it by hygienic and dietetic measures. If drugs must be used they should be the milder saline laxatives or the compound liquorice powder. Enemata are often necessary, and it is much preferable to employ them early than to constantly use purgative pills. Glycerine either in the form of suppository or as a small injection is very valuable. Half a drachm of boric acid placed within the rectum is sometimes efficacious. The injections of tepid water, with or without soap, may be used for a prolonged period with good effect and without damage. The patient should be in the

dorsal position with the hips elevated, and it is best to let the fluid flow in slowly from a fountain syringe.

There are various drugs which are of special service, particularly the combination of ipecacuanha, nux vomica, or belladonna, with aloes, rhubarb, colocynth, or podophyllin. Meigs recommends particularly the combination of extract of belladonna (gr. $\frac{1}{12}$), extract of nux vomica (gr. $\frac{1}{4}$), and extract of colocynth (gr. ij), one pill to be taken three times a day. In anæmia and chlorosis a sulphur confection taken in the morning, and a pill of iron, rhubarb, and aloes throughout the day are very serviceable.

In children the indications should be met, as far as possible, by hygienic and dietetic measures. In the constipation of sucklings a change in the diet of the mother may be tried. Drinking of water, barley water, or oatmeal water will sometimes obviate the difficulty. If laxatives are required simple syrup, manna, or olive oil may be sufficient. The conical piece of soap, so often seen in nurseries, is sometimes efficacious. Small injections of cold water may be used. Large injections should be avoided if possible. If it is necessary to give a laxative by the mouth the castor oil or fluid magnesia is the best. If there are signs of gastro-intestinal irritation rhubarb and soda or gray powder may be given. In older children the diet should be carefully regulated.

VIII. DISEASES OF THE LIVER.

I. JAUNDICE (*Icterus*).

1. **Jaundice as a Symptom.**—Cases with icterus may be divided into two great groups: Those in which there is obstruction, either in the smaller or in the larger ducts—the *hepatogenous* form; cases in which the jaundice is due to suppression of the function of the liver-cells, as in the widespread necrosis of acute yellow atrophy, or to an excess of the chromatogenous material, as in malaria, pernicious anæmia, and certain fevers, in which the liver function cannot keep pace with the blood destruction (hæmolysis)—*hæmatogenous* or non-obstructive jaundice.

The following classification of the causes of hepatogenous jaundice is arranged by Murchison, to whose writings on the liver we owe so much: Obstruction (1) by foreign bodies within the ducts, as gall-stones and parasites; (2) by inflammatory tumefaction of the duodenum or of the lining membrane of the duct; (3) by stricture or obliteration of the duct; (4) by tumors closing the orifice of the duct or growing in its interior; (5) by pressure on the duct from without, as by tumors of the liver itself, of the stomach, pancreas, kidney, or omentum; by pressure of enlarged glands in the fissure of the liver, and, more rarely, of abdominal aneurism,

fæcal accumulation, or the pregnant uterus; (6) to these may be added lowering of the blood pressure in the liver, so that the tension in the smaller bile-ducts is greater than in the blood-vessels. In this class very probably may be placed the cases resulting from mental shock or depressing emotions.

General Symptoms of Obstructive Jaundice.—(1) Icterus, or tinting of the skin and conjunctivæ. The color ranges from a lemon-yellow in catarrhal jaundice to a deep olive-green or bronzed hue in permanent obstruction. In some instances the color of the skin is greenish black, the so-called "black jaundice."

(2) Of other cutaneous symptoms, pruritus in the more chronic forms may be intense and cause the greatest distress. It may precede the onset of the jaundice, but as a rule it is not very marked except in cases of prolonged obstruction. Sweating is common, and may be curiously localized to the abdomen or to the palms of the hands. Lichen, urticaria, and boils may develop, and the skin disease known as xanthelasma or vitiligoidea.

(3) The secretions are colored with bile-pigment. The sweat tinges the linen; the tears and saliva and milk are rarely stained. The expectoration is not often tinted unless there is inflammation, as when pneumonia coexists with jaundice. The urine may contain the pigment before it is apparent in the skin or conjunctiva. The color varies from light greenish yellow to a deep black-green. Gmelin's test is made by allowing five or six drops of urine and a similar amount of common nitric acid to flow together slowly on the flat surface of a white plate. A play of colors is produced—various shades of green, yellow, violet, and red. In cases of jaundice of long standing or great intensity the urine usually contains albumen and always bile-stained tube-casts.

(4) No bile passes into the intestine. The stools therefore are of a pale drab or slate-gray color, and usually very fetid and pasty. There may be constipation; in many instances, owing to decomposition, there is diarrhœa.

(5) Slow pulse. The heart's action may fall to 40, 30, or even to 20 per minute. It is particularly noticeable in the cases of catarrhal jaundice, and is not as a rule an unfavorable symptom.

(6) Hæmorrhage. Ecchymoses are not uncommon in severe jaundice, particularly in the more malignant forms.

(7) Cerebral symptoms. Irritability, great depression of spirits, or even melancholia may be present. In any case of persistent jaundice special nervous phenomena may develop and rapidly prove fatal—such as sudden coma, acute delirium or convulsions. Usually the patient has a rapid pulse, slight fever, and a dry tongue, and he passes into the so-called "typhoid state." These features are not nearly so common in obstructive as in febrile jaundice, but they not infrequently terminate a chronic icterus in whatever way produced. The group of symptoms has been termed

cholæmia or, on the supposition that cholesterin is the poison, *cholesteræmia;* but the true nature of the poison has not yet been determined. In some of the cases the symptoms may be due to uræmia.

Non-obstructive jaundice may be thus classified:

(1) The form in which there is wide-spread necrosis of the liver-cells and direct interference with their bile-forming function, as in acute yellow atrophy, and possibly in certain cases of hypertrophic cirrhosis. Strictly speaking, this is a hepatogenous jaundice.

(2) The toxic form. The poisons of yellow fever, malaria, typhoid, epidemic jaundice, and pyæmia; snake virus, as well as chloroform, ether, phosphorus, and mercury, act by causing increased destruction of the red blood-corpuscles. More blood-pigment is set free than can be disposed of by liver, spleen, or kidneys, and the bilirubin (transformed hæmoglobin) is deposited in the tissues. The *symptoms* of hæmatogenous jaundice are not nearly so striking as in the obstructive variety. The skin has in many cases only a light lemon tint. In the severer forms, as in acute yellow atrophy, the color may be more intense, but in malaria and pernicious anæmia the tint is usually light. In these mild cases the urine may contain little or no bile-pigment, but the urinary pigments are considerably increased. The stools are not clay-colored and may in some instances be very dark. In the toxic forms of this variety the cerebral symptoms are marked and there may be active delirium, coma, or convulsions.

2. **Icterus Neonatorum.**—New-born infants are liable to jaundice, which in some instances rapidly proves fatal. A mild and a severe form may be recognized.

The *mild icterus* of the new-born is a common disease in foundling hospitals and is not very infrequent in private practice. The discoloration appears early, usually on the first or second day, and is of moderate intensity. The urine may be bile-stained and the fæces colorless. The nutrition of the child is not seriously disturbed, and in the majority of cases the jaundice disappears within two weeks. It is supposed that the diminished pressure in the portal vessels, following the severance of the placental circulation, allows absorption from the bile capillaries, in which the tension is greater. Possibly too, as Quincke suggests, the ductus venosus may remain open, allowing some of the portal blood containing bile to flow into the systemic circulation. On the other hand, it is held that the jaundice is hæmatogenous and due to the destruction of large numbers of red blood-corpuscles during the first few days after birth.

The *severe form* of icterus in the new-born may depend upon (*a*) congenital absence of the common or hepatic duct of which there are several instances on record; (*b*) congenital syphilitic hepatitis; and (*c*) septic poisoning, associated with phlebitis of the umbilical vein. This is a severe and fatal form, in which also hæmorrhage from the cord may occur.

Occasionally jaundice sets in and persists for many weeks, or even months, without interfering seriously with the nutrition of the child.

3. Acute Yellow Atrophy of the Liver; Malignant Jaundice; Icterus Gravis.

Definition.—Jaundice associated with marked cerebral symptoms and characterized anatomically by extensive necrosis of the liver-cells with reduction in volume of the organ.

Etiology.—This is a rare disease. In a somewhat varied post-mortem and clinical experience no instance has fallen under my observation. On the other hand, a physician may see several cases within a few years, or even within a few months, as happened to Riess, who saw five cases within three months at the Charité, in Berlin. The disease seems to be rare in this country. No case is reported in the Transactions of the Pathological Societies of New York (Vols. I to III) or of Philadelphia (Vols. I to XIII). The disease is more common in women than in men. Of the 100 cases collected by Legg, 69 were in females; and of Thierfelder's 143 cases, 88 were in women. There is a remarkable association between the disease and pregnancy, which was present in 25 of the 69 women in Legg's statistics, and in 33 of the 88 women in Thierfelder's collection. It is most common between the ages of twenty and thirty, but is occasionally seen in young children. It has followed fright or profound mental emotion. Though the symptoms produced by phosphorus poisoning closely simulate those of acute yellow atrophy, the two conditions are not identical.

Morbid Anatomy.—The liver is greatly reduced in size, looks thin and flattened, and sometimes does not reach more than one half or even one third of its normal weight. It is flabby and the capsule is wrinkled. On section the color is of a yellowish brown, yellowish red, or mottled, and the outlines of the lobules are indistinct. The yellow and dark-red portions represent different stages of the same process—the yellow an earlier, the red a more advanced stage. The organ may cut with considerable firmness. Microscopically the liver-cells are seen in all stages of necrosis, and in spots appear to have undergone complete destruction, leaving a fatty, granular *débris* with pigment grains and crystals of leucin and tyrosin. The interlobular tissue may be normal, but in many cases there is a marked proliferation of small cells, which was present in 9 of the 12 cases examined by Riess. Micro-organisms have been noted by several observers. The bile-ducts and gall-bladder are empty.

The other organs show extensive bile staining, and there are numerous hæmorrhages. The kidneys may show marked granular degeneration of the epithelium, and usually there is fatty degeneration of the heart. In a majority of the cases the spleen is enlarged.

Symptoms.—In the initial stage there is a gastro-duodenal catarrh, and at first the jaundice is thought to be of a simple nature. In some in-

stances this lasts only a few days, in others two or three weeks. Then severe symptoms set in—headache, delirium, trembling of the muscles, and, in some instances, convulsions. Vomiting is a constant symptom, and blood may be brought up. Hæmorrhages occur into the skin or from the mucous surfaces; in pregnant women abortion may occur. With the development of the head symptoms the jaundice usually increases. Coma sets in and gradually deepens until death. The body temperature is variable; in a majority of the cases the disease runs an afebrile course, though sometimes just before death there is an elevation. In some instances, however, there has been marked pyrexia. The pulse is usually rapid, the tongue coated and dry, and the patient is in a "typhoid state."

The urine is bile-stained and often contains tube-casts. Leucin and tyrosin are constantly present; the former as rounded disks, the latter in needle-shaped crystals, arranged either in bundles or in groups. The tyrosin may sometimes be seen in the urine sediment, but it is best first to evaporate a few drops of urine on a cover-glass. In the majority of cases no bile enters the intestines, and the stools are clay-colored. The disease is almost invariably fatal. In a few instances recovery has been noted. I saw in Leube's clinic, at Wurzburg, a case which was convalescent.

Diagnosis.—Jaundice with delirium, diminution of the liver volume, delirium, and the presence of leucin and tyrosin in the urine, form a characteristic and unmistakable group of symptoms.

It is not to be forgotten that any severe jaundice may be associated with intense cerebral symptoms. The clinical features in certain cases of hypertrophic cirrhosis are almost identical, but the enlargement of the liver, the more constant occurrence of fever, and the absence of leucin and tyrosin are distinguishing signs. Phosphorus poisoning may closely simulate acute yellow atrophy, particularly in the hæmorrhages, jaundice, and the diminution in the liver volume, but the gastric symptoms are usually more marked, and leucin and tyrosin are stated not to occur in the urine.

No known remedies have any influence on the course of the disease.

II. AFFECTIONS OF THE BLOOD-VESSELS OF THE LIVER.

(1) **Anæmia.**—On the post-mortem table, when the liver looks anæmic, as in the fatty or amyloid organ, the blood-vessels, which during life were probably well filled, can be readily injected. There are no symptoms indicative of this condition.

(2) **Hyperæmia.**—This occurs in two forms. (*a*) *Active hyperæmia.* After each meal the rapid absorption by the portal vessels induces transient congestion of the organ, which, however, is entirely physiological; but it is quite possible that in persons who persistently eat and drink too much

this active hyperæmia may lead to functional disturbance or, in the case of drinking too freely of alcohol, to organic change.

The *symptoms* of active hyperæmia are indefinite. Possibly the sense of distress or fulness in the right hypochondrium, so often mentioned by dyspeptics and by those who eat and drink freely, may be due to this cause. There are probably diurnal variations in the volume of the liver. In cirrhosis with enlargement the rapid reduction in volume after a copious hæmorrhage indicates the important part which hyperæmia plays even in organic troubles. It is stated that suppression of the menses or suppression of a hæmorrhoidal flow is followed by hyperæmia of the liver. Andrew H. Smith has described a case of periodical enlargement of the liver.

(*b*) *Passive Congestion.*—This is much more common and results from an increase of pressure in the efferent vessels or sub-lobular branches of the hepatic veins. Every condition leading to venous stasis in the right heart at once affects these veins.

In chronic valvular disease, in emphysema, cirrhosis of the lung, and in intrathoracic tumors mechanical congestion occurs and finally leads to very definite changes. The liver is enlarged, firm, and of a deep-red color; the hepatic vessels are greatly engorged, particularly the central vein in each lobule and its adjacent capillaries. On section the organ presents a peculiar mottled appearance, owing to the deeply congested hepatic and the anæmic portal territories; hence the term *nutmeg* which has been given to this condition. Gradually the distention of the central capillaries reaches such a grade that atrophy of the intervening liver-cells is induced. Brown pigment is deposited about the centre of the lobules and the connective tissue is greatly increased. In this cyanotic induration or cardiac liver the organ is large in the early stage, but later it may become contracted. Occasionally in this form the connective tissue is increased about the lobules as well, but the process usually extends from the sublobular and central veins.

The symptoms of this form are not always to be separated from those of the associated conditions. Gastro-intestinal catarrh is usually present and hæmatemesis may occur. The portal obstruction in advanced cases leads to ascites, which may precede the development of general dropsy. There is often slight jaundice, the stools may be clay-colored, and the urine contains bile-pigment.

On examination the organ is found to be increased in size. It may be a full hand's-breadth below the costal margin and tender on pressure. It is in this condition particularly that we meet with pulsation of the liver. We must distinguish the communicated throbbing of the heart, which is very common, from the heaving, diffuse impulse due to regurgitation into the hepatic veins, in which, when one hand is upon the ensiform cartilage and the other upon the right side at the margin of the ribs, the whole liver can be felt to dilate with each impulse.

The indications for *treatment* in passive hyperæmia are to restore the balance of the circulation and to unload the engorged portal vessels. In cases of intense hyperæmia eighteen or twenty ounces of blood may be directly aspirated from the liver, as advised by George Harley and practised by many Anglo-Indian physicians. Good results sometimes follow this hepato-phlebotomy. The prompt relief and marked reduction in the volume of the organ which follow an attack of hæmatemesis or bleeding from piles suggests this practice. Salts administered by Matthew Hay's method deplete the portal system freely and thoroughly. As a rule, the treatment must be that of the condition with which it is associated.

(3) **Diseases of the Portal Vein.**—(*a*) *Thrombosis; Adhesive Pylephlebitis.*—Coagulation of blood in the portal vein is rarely seen except in cirrhosis. Exceptional causes are invasion of the branches by cancer, proliferative peritonitis involving the gastro-hepatic omentum, and perforation of the vein by gall-stones. In rare instances a complete collateral circulation is established, the thrombus undergoes the usual changes, and ultimately the vein is represented by a fibrous cord, a condition which has been called *pylephlebitis adhesiva.* In a case of this kind which I dissected the portal vein was represented by a narrow fibrous cord; the collateral circulation, which must have been completely established for years, ultimately failed, ascites and hæmatemesis supervened and rapidly proved fatal.* The diagnosis of obstruction of the portal vein can rarely be made. A suggestive symptom, however, is a *sudden* onset of the most intense engorgement of the branches of the portal system.

Emboli in the branches of the portal vein do not, as a rule, produce infarction, for blood reaches the lobular capillary plexus, as shown by Cohnheim and Litten, through the free anastomosis with the hepatic artery. In rare instances, however, a condition resembling infarction does occur, sometimes in small areas, at others in quite extensive territories. Septic emboli, on the other hand, may induce suppuration.

(*b*) *Suppurative pylephlebitis* will be considered in the section on abscess.

(4) **Affections of the hepatic vein** are extremely rare. Dilatation occurs in cases of chronic enlargement of the right heart, from whatever cause produced. Emboli occasionally pass from the right auricle into the hepatic veins. A rare and unusual event is stenosis of the orifices of the hepatic veins, which I met in a case of fibroid obliteration of the inferior vena cava and was associated with a greatly enlarged and indurated liver.†

(5) **Hepatic Artery.**—Enlargement of this vessel is seen in cases of cirrhosis of the liver. It may be the seat of extensive sclerosis. Aneurism of the hepatic artery is rare, but instances are on record, and will be referred to in the section on arteries.

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* Journal of Anatomy and Physiology, vol. xvii.

† Ibid., vol. xvi.

III. DISEASES OF THE BILE-PASSAGES.

Catarrhal Jaundice.

Definition.—Jaundice due to swelling and obstruction of the terminal portion of the common duct.

Etiology.—General catarrhal inflammation of the bile-ducts is usually associated with gall-stones. The catarrhal process now under consideration is probably always an extension of a gastro-duodenal catarrh, and the process is most intense in the *pars intestinalis* of the duct, which projects into the duodenum. The mucous membrane is swollen, and a plug of inspissated mucus fills the diverticulum of Vater, and the narrower portion just at the orifice, completely obstructing the outflow of bile. It is not known how wide-spread this catarrh is in the bile-passages, and whether it really passes up the ducts. It would, of course, be possible to have a catarrh of the finer ducts within the liver, which some French writers think may initiate the attack, but the evidence of this is not strong, and it seems more likely that the terminal portion of the duct is always first involved. In the only instance which I have had an opportunity to examine post mortem the orifice was plugged with inspissated mucus, the common and hepatic ducts were slightly distended and contained a bile-tinged, not a clear, mucus, and there were no observable changes in the mucosa of the ducts.

This catarrhal or simple jaundice results from the following causes: (1) Duodenal catarrh, in whatever way produced, most commonly following an attack of indigestion. It is most frequently met with in young persons, but may occur at any age, and may follow not only errors in diet, but also cold, exposure, and malaria, as well as the conditions associated with portal obstruction, chronic heart-disease, and Bright's disease. (2) Emotional disturbances may be followed by jaundice, which is believed to be due to catarrhal swelling. Cases of this kind are rare and the anatomical condition is unknown. (3) Simple or catarrhal jaundice may occur in epidemic form. (4) Catarrhal jaundice is occasionally seen in the infectious fevers, such as pneumonia, and typhoid fever.

Symptoms.—There may be neither pain nor distress, and the patient's friends may first notice the yellow tint, or the patient himself may observe it in the looking-glass. In other instances there are dyspeptic symptoms and uneasy sensations in the hepatic region or pains in the back and limbs. In the epidemic form, the onset may be more severe, with headache, chill, and vomiting. Fever is rarely present, though the temperature may reach 101°, sometimes 102°. All the signs of obstructive jaundice already mentioned are present, the stools are clay-colored, and the urine contains bile-pigment. The jaundice has a bright-yellow tint; the greenish, bronzed color is never seen in the simple form. The pulse may be normal, but occasionally it is remarkably slow, and may fall

to forty or thirty beats in the minute. The liver may be normal in size, but is usually slightly enlarged, and the edge can be felt below the costal margin. Occasionally the enlargement is more marked. The duration of the disease is from four to eight weeks. There are mild cases in which the jaundice disappears within two weeks; on the other hand, it may persist for three months. The stools should be carefully watched, for they give the first intimation of removal of the obstruction.

The *diagnosis* is rarely difficult. The onset in young, comparatively healthy persons, the moderate grade of icterus, the absence of emaciation or of evidences of cirrhosis or cancer, usually make the diagnosis easy. Cases which persist for two and three months cause uneasiness, as the suspicion is aroused that it may be more than simple catarrh. The absence of pain, the negative character of the physical examination, and the maintenance of the general nutrition are the points in favor of simple jaundice. There are instances in which time alone can determine the true nature of the case.

Treatment.—As a rule the patient can keep on his feet from the outset. Measures should be used to allay the gastric catarrh, if it is present. A dose of calomel may be given, and the bowels kept open subsequently by salines. The patient should not be violently purged. Bismuth and bicarbonate of soda may be given, and the patient should drink freely of the alkaline mineral waters, of which Vichy is the best. Irrigation of the large bowel with cold water may be practised. The cold is supposed to excite peristalsis of the gall-bladder and ducts, and thus aid in the expulsion of the mucus. This practice has been followed in my wards for several years, but I cannot speak warmly of the results.

Cholelithiasis (*Gall-Stones*).

Calculi are formed in the gall-bladder. Evidence is wanting to show that they are formed within the liver ducts, except in very rare instances. They may be single, in which case the stone is usually ovoid and may attain a very large size. Instances are on record of gall-stones measuring more than five inches in length. They may be extremely numerous, ranging from a score to several hundreds or even several thousands, in which case the stones are very small. When moderately numerous, they show signs of mutual pressure and have a polygonal form, with smooth facets; occasionally, however, five or six gall-stones of medium size are met with in the bladder which are round or ovoid and without facets. They are sometimes mulberry-shaped and very dark, consisting largely of bile-pigment. Again there are small, black calculi, rough and irregular in shape, and varying in size from sand to small shot. These are sometimes known as gall-sand. On section, a calculus contains a nucleus, which consists of bile-pigment, rarely a foreign body. The greater portion of the stone is made up of cholesterin, which may form the entire calculus and is ar-

ranged in concentric laminæ showing also radiating lines. Salts of lime and magnesia, bile acids, fatty acids, and traces of iron and copper are also found in them. A majority of gall-stones consist of from seventy to eighty per cent of cholesterin, in either the amorphous or the crystalline form. As above stated, it is sometimes pure, but more commonly it is mixed with the bile-pigment. The outer layer of the stone is usually harder and brownish in color, and contains a larger proportion of lime salts.

The mode of formation is by no means clear. A defect in the sodium salts seems to favor the precipitation of the cholesterin and of the bile-pigment. The lime exists in such slight quantities in the bile that it is probably a pathological product of the mucous glands of the gall-bladder. When the bile is retained long in the gall-bladder its concentration favors the deposition.

Etiology.—Three fourths of the cases of gall-stones occur in women, most frequently between the ages of thirty and sixty. Sedentary occupations, particularly when combined with overindulgence in eating, seem important factors. The subjects are often stout, and usually very fond of starchy and saccharine food. The conditions which induce lithic acid also favor the development of gall-stones. Tight-lacing is regarded by Marchand as an important factor in retarding the flow of the bile. Pregnancy has a similar influence. Naunyn states that ninety per cent of women with gall-stones have borne children. Constipation and depressing mental influences have been regarded as favoring circumstances.

Symptoms.—In a majority of the cases, gall-stones cause no symptoms. The gall-bladder will tolerate the presence of large numbers for an indefinite period of time, and post-mortem examinations show that they are present in twenty-five per cent of all women over sixty years of age (Naunyn).

The effects of gall-stones may be considered under the following headings: The symptoms produced by the passage of a stone through the ducts—biliary colic; the effects of permanent plugging of the duct; and the more remote effects, due to ulceration and perforation, and the establishment of fistulæ.

1. **Biliary Colic.**—It would appear that gall-stones may become engaged in the cystic or the common duct without producing pain or severe symptoms. More commonly the passage of a stone excites the violent symptoms known as biliary colic. The attack sets in abruptly with agonizing pain in the right hypochondriac region, which radiates to the shoulder, or is very intense in the epigastric and in the lower thoracic regions. It is often associated with a rigor and a rise in temperature from 102° to 103°. The pain is usually so intense that the patient rolls about in agony. There are vomiting, profuse sweating, and great depression of the circulation. There may be marked tenderness in the region of the liver, which may become enlarged. In a large number of the cases jaundice

develops, but it is not a necessary symptom. Of course it does not occur during the passage of the stone through the cystic duct, but only when it becomes lodged in the common duct. Probably the intense pain is due to the slow progress in the cystic duct, in which the stone takes a rotary course owing to the arrangement of the Heisterian valve.

The attack varies in duration. It may last for a few hours, several days, or even a week or more. If the stone becomes impacted in the orifice of the common duct, the jaundice becomes intense; much more commonly it is a slight, transient icterus. The attack of colic may be repeated at intervals for some time, but finally the stone passes and the symptoms rapidly disappear.

Occasionally accidents occur, such as rupture of the duct with fatal peritonitis. Syncope, owing to the intensity of the pain, may follow and has been known to prove fatal, and epilepsy has been seen. These are, however, rare events. Palpitation and distress about the heart may be present, and occasionally a mitral murmur develops during the paroxysm; but the cardiac conditions described by some writers as coming on acutely in biliary colic are probably pre-existent in these patients.

The *diagnosis* of acute hepatic colic is generally easy. The pain is in the upper abdominal and thoracic regions, whereas the pain in nephritic colic is in the lower abdomen. A chill, with fever, is much more frequent in biliary colic than in gastralgia, with which it is liable, at times, to be confounded. A history of previous attacks is an important guide, and the occurrence of jaundice, however slight, determines the diagnosis. To look for the gall-stones, the stools should be thoroughly mixed with water and carefully filtered through a narrow-meshed sieve.

2. **Chronic Obstruction of the Ducts by Gall-stones.**—*Of the Cystic Duct.*—The effects may be thus enumerated:

Dilatation of the gall-bladder—hydrops vesicæ felleæ. This occurs much more frequently than in obstruction of the common duct. The fluid is almost invariably of a thin mucoid nature, though it may be mixed with bile. In all cases, when the obstruction persists, the bile is replaced by a clear fluid. This is an important point in diagnosis, particularly as a dropsical gall-bladder may form a very large tumor. The reaction is not always constant. It is either alkaline or neutral; the consistence is thin and mucoid. Albumen is usually present. The organ may reach an enormous size, and in one instance Tait found it occupying the greater part of the abdomen. In such cases, as is not unnatural, it has been mistaken for an ovarian tumor. In one of my cases it was adherent to the broad ligament, and had been mistaken for a cyst of the left ovary. The dilated gall-bladder can usually be felt below the edge of the liver, and in many instances it has a characteristic outline like a gourd. It usually projects directly downward, rarely to one side or the other, though occasionally toward the middle line. It may reach below the navel, and in persons with thin walls the outline can be accurately

defined. It is to be remembered that distention of the gall-bladder may occur without jaundice; indeed, the greatest enlargement has been met with in such cases.

In obstruction of the common duct the gall-bladder is not necessarily greatly enlarged. Occasionally it may be much distended without the occurrence of any tumor which can be felt during life. In one case (operation) eighteen ounces were removed from a gall-bladder, the edge of which barely projected below the margin of the right lobe.

Acute phlegmonous cystitis. This is a rare event. Only seven instances of it have been collected in the enormous statistics of Courvoisier. In a case which I have reported the patient died on the fifth day with symptoms of the most intense prostration, fever, and abdominal pain. Perforation may occur with fatal peritonitis.

Suppurative cholecystitis, empyema of the gall-bladder, is much more common, and in the great majority of cases is associated with gall-stones —41 in 55 cases (Courvoisier). There may be enormous dilatation, and over a litre of pus has been found. Perforation and the formation of abscesses in the neighborhood are not uncommon.

Calcification of the gall-bladder is commonly a termination of the previous condition. There are two separate forms, incrustation of the mucosa with lime salts and the true infiltration of the wall with lime, the so-called ossification. A remarkable example of the latter sent to me by Groves, of Carp, is now in the McGill Medical Museum.

Atrophy of the gall-bladder. This is by no means uncommon. The organ shrinks into a small fibroid mass, not larger, perhaps, than a good-sized pea or walnut, or even has the form of a narrow fibrous string; more commonly the gall-bladder tightly embraces a stone. This condition is usually preceded by hydrops of the bladder. In an interesting case of the kind, the patient, nearly twenty years before, had had an obscure abdominal tumor, which caused so much difference of opinion among his physicians that instruction was left in his will that the body should be examined. The gall-bladder was entirely obliterated and closely encircled a large gall-stone.

Occasionally the gall-bladder presents diverticula, which may be cut off from the main portion, and usually contain calculi.

Obstruction of the Common Duct.

The stone usually lies at the termination of the duct, just at the orifice of the papilla, within a sort of pouch formed by the diverticulum of Vater. Examined from the duodenum, it seems to be directly beneath the mucosa. It is as a rule single; but two and, in some instances, a series of stones may occupy the entire duct. The effect of the obstruction is dilatation, with catarrhal or suppurative cholangitis.

(1) Obstruction, with catarrhal cholangitis.

The common duct may be as large as the thumb; the hepatic duct and its branches through the liver are greatly dilated, and the distention may

even be apparent beneath the liver capsule. Great enlargement of the gall-bladder is rare. The mucous membrane of the ducts may be smooth and clear, and the contents a thin, colorless mucus.

Catarrhal cholangitis with gall-stones is characterized by a special symptom group: (*a*) Ague-like paroxysms, chills, fever, and sweating; (*b*) jaundice of varying intensity, which persists for months or even years, and deepens after each paroxysm; (*c*) at the time of the paroxysms, pains in the region of the liver with gastric disturbance. These symptoms may continue on and off for three or four years, without the development of suppurative cholangitis. In one of my cases the jaundice and recurring hepatic intermittent fever existed from July, 1879, until August, 1882; the patient recovered and still lives. The condition has lasted from eight months to three years. The rigors are of intense severity, and the temperature rises to 103° or 105°. The chills may recur daily for weeks, and present a tertian or quartan type, so that they often are mistaken for malaria, with which, however, they have no connection. The jaundice is variable, and deepens after each paroxysm. Pain, which is sometimes intense and colicky, does not always occur. There may be marked vomiting and nausea. As a rule there is no progressive deterioration of health. In the intervals between the attacks the temperature is normal.

The clinical history and the post-mortem examinations in my cases* have shown conclusively that this condition may persist for years without a trace of suppuration within the ducts.

The nature of the hepatic intermittent fever is not settled. Charcot holds that it is due to the production of a ferment in the bile-passages, and a bacillus, probably the *bacterium coli commune*, has been found in the ducts in several cases. Both Murchison and Ord hold that it is simply due to local irritation of the mucous membrane, and that the fever is really of a nervous character.

The effect upon the liver of chronic obstruction of the bile-duct is very variable. The organ is rarely enlarged. It is firm and the connective tissue is moderately increased. In none of my cases of persistent obstruction by gall-stones was the liver greatly enlarged, nor did it present macroscopically the features of cirrhosis. On this point my experience is in accord with that of Sharkey, who has recently called in question the statements of Charcot and Wickham Legg as to the occurrence of cirrhosis under these circumstances.

(2) Obstruction, with suppurative cholangitis.

When suppurative cholangitis exists the mucosa is thickened, often eroded or ulcerated; there may be extensive suppuration in the ducts throughout the liver, and even empyema of the gall-bladder. Occasionally

* On Fever of Hepatic Origin, particularly the Intermittent Pyrexia associated with Gall-stones, Johns Hopkins Hospital Reports, vol. ii, No. 1, 1890; and in Annals of Surgery, 1890.

the suppuration extends beyond the ducts, and there is localized liver abscess, or there is perforation of the gall-bladder with the formation of abscess between the liver and stomach.

Clinically it is characterized by a fever which may be intermittent, but more commonly is remittent and without prolonged intervals of apyrexia. The jaundice is rarely so intense, nor do we see the deepening of the color after the paroxysms. There is usually greater enlargement of the liver and tenderness and more definite signs of septicæmia. The cases run a shorter course, and recovery never takes place.

3. **The More Remote Effects of Gall-stones.** — (*a*) Biliary Fistulæ. These are not uncommon. There may, for instance, be abnormal communication between the gall-bladder and the hepatic duct or the gall-bladder and a cavity in the liver itself. More rarely perforation occurs between the common duct and the portal vein. Of this there are at least four instances on record, among them the celebrated case of Ignatius Loyola. Perforation into the abdominal cavity is not uncommon; 119 cases exist in the literature (Courvoisier), in 70 of which the rupture occurred directly into the peritoneal cavity; in 49 there was encapsulated abscess. Perforation may take place from an intrahepatic branch or from the hepatic, common, or cystic ducts. Perforation from the gall-bladder is the most common.

Fistulous communications between the bile-passages and the gastro-intestinal canal are frequent. Openings into the stomach are rare. Between the duodenum and bile-passages they are much more common. Courvoisier has collected 10 instances of communication between the ductus communis and the duodenum, and 73 cases between the gall-bladder and the duodenum. Communication with the ileum and jejunum is extremely rare. Of fistulous opening into the colon 39 cases are on record. These communications can rarely be diagnosed; they may be present without any symptoms whatever. It is probably by ulceration into the duodenum or colon that the large gall-stones escape.

Occasionally fistulous communication exists between the gall-bladder and the urinary passages, and the stones may be found in the bladder. The opening has been either into the pelvis of the kidney or, as has been supposed, the gall-bladder has become adherent in the neighborhood of the navel, and the stone has escaped through an open urachus. It is possible that adhesions may form between the distended gall-bladder and urinary bladder, since the former has been found adherent as low as the broad ligament.

Many instances are on record of fistulæ between the bile-passages and the lungs. Courvoisier has collected twenty-four cases. Bile may be coughed up with the expectoration, sometimes in considerable quantities. In only seven cases did recovery take place. In some of these the abscess formation was due to hydatids, in some to ascarides. The perforation usually takes place through the lung, by a liver abscess communicating with

the pleura, or occasionally the abscess enters the mediastinum and perforates a bronchus.

Of all fistulous communications the external or cutaneous is the most common. Courvoisier's statistics number 184 cases, in fifty per cent of which the perforation took place in the right hypochondrium; in twenty-nine per cent in the region of the navel. The number of stones discharged varied from one or two to many hundreds. Recovery took place in 78 cases; some with, some without operation.

(*b*) Obstruction of the bowel by gall-stones. Reference has already been made to this, the frequency which appears from the fact that of 295 cases of obstruction, occurring during the past eight years, analyzed by Fitz, 23 were by gall-stone. Courvoisier's statistics give a total number of 131 cases, in six of which the calculi had a peculiar situation, as in a diverticulum or in the appendix. Of the remaining 125 cases, in 70 the stone was spontaneously passed, usually with severe symptoms. The post-mortem reports show that in some of these cases even very large stones have passed *per viam naturalem*, as the gall-duct has been enormously distended, its orifice admitting the finger freely. This, however, is extremely rare. The stones have been found most commonly in the ileum.

Other Affections of the Bile-ducts.

Cancer will be considered later.

Stenosis or complete occlusion may follow ulceration, most commonly after the passage of a gall-stone. In these instances the obstruction is usually situated low down in the common duct. Instances of this are extremely rare. Foreign bodies, such as the seeds of various fruits, may enter the duct, and occasionally round worms crawl into it. In the Wistar-Horner Museum of the University of Pennsylvania there is a remarkable specimen showing the common and hepatic ducts enormously distended and densely packed with a dozen or more lumbricoid worms. A similar specimen exists in one of the Paris museums. Liver-flukes and echinococci are rare causes of obstruction in man.

Obstruction by *pressure* from without is more frequent. Naturally cancer of the head of the pancreas is apt to involve the terminal portion of the duct; less often cancer of the pylorus. Secondary involvement of the lymph glands of the liver is a common cause of occlusion of the duct, and is met with in many cases of cancer of the stomach and other abdominal organs. Rare causes of obstruction are aneurism of a branch of the cœliac axis or of the aorta, or pressure of very large abdominal tumors.

The symptoms produced are those of chronic obstructive jaundice. At first, the liver is usually enlarged, but in chronic cases it may be reduced in size, and of a deeply bronzed color, and firm, owing to slight increase in the connective tissue. The hepatic intermittent fever may be associated with occlusion of the duct from any cause, but it is most fre-

quently met with in chronic obstruction by gall-stones. Permanent occlusion of the duct terminates in death. In a majority of the cases the conditions which lead to the obstruction are in themselves fatal. Cases of cicatricial occlusion may last for years. A patient under my care, who was permanently jaundiced for nearly three years, had a fibroid occlusion of the duct.

The *diagnosis* of the nature of the occlusion is often very difficult. A history of colic, jaundice of varying intensity, paroxysms of pain, and intermittent fever point to gall-stones. In cancerous obstruction the tumor mass can sometimes be felt in the epigastric region. In cases in which the lymph glands in the transverse fissure are cancerous, the primary disease may be in the pelvic organs or the rectum, or there may be a limited cancer of the stomach, which has not given any symptoms. In these cases the examination of the other lymphatic glands may be of value. In a case, recently under observation, with jaundice of seven weeks' duration, and believed to be catarrhal (as the patient's general condition was good and he was said not to have lost flesh), a small nodular mass was detected at the navel, which on removal proved to be scirrhus. Involvement of the clavicular groups of lymph glands may also be serviceable in diagnosis. As already mentioned, the gall-bladder is often but little enlarged in obstruction of the common duct. Great and progressive enlargement of the liver with jaundice and moderate continued fever is more commonly met with in cancer. In hypertrophic cirrhosis a similar condition exists, but the organ is smooth and there is rarely progressive enlargement while under observation.

Treatment of Gall-stones and their Effects.—In an attack of biliary colic the patient should be kept under morphia, given hypodermically, in quarter-grain doses. In an agonizing paroxysm it is well to give a whiff or two of chloroform until the morphia has had time to act. Great relief is experienced from the hot bath and from fomentations in the region of the liver. The patient should be given laxatives and should drink copiously of alkaline mineral waters. Olive oil has proved useless in my hands. When taken in large quantities, fatty concretions are passed with the stools, which have been mistaken for calculi. Since the days of Durande, whose mixture of ether and turpentine is still largely used in France, various remedies have been advised to dissolve the stones within the gall-bladder, none of which are efficacious.

The diet should be regulated, the patient should take regular exercise and avoid, as much as possible, the starchy and saccharine foods. The soda salts recommended by Prout are believed to prevent the concentration of the bile and the formation of gall-stones. Either the sulphate or the phosphate may be taken in doses of from one to two drachms daily.

Expression of gall-stones from the bladder by digital manipulation, as recommended by George Harley, is a highly irrational procedure, not to be followed. So long as gall-stones remain in the bladder they do little

or no harm in a great majority of cases. To force them on into the duct is to render the patient liable to severe colic or to the still more serious danger of permanent obstruction.

When the cystic duct is occluded and the gall-bladder distended, an exploratory puncture may be made, as practised by the elder Pepper, in 1857, in a case of empyema of the gall-bladder, and by Bartholow in 1878. The puncture may be made either to draw off fluid from a distended bladder or to explore for gall-stones. Aspiration is usually a safe procedure, though a fatal result has followed. When the gall-bladder is distended and plainly palpable, to sound for stones by an exploratory puncture is justifiable, but under no other circumstances. "The easy and safe method of sounding for impacted stones," recommended a few years ago by a London physician, in which it is advised to thrust a sharp needle six inches long between the navel and the margin of the liver, may be characterized as one of the most extraordinary operations ever advocated, and would probably always prove fatal, as in the case of the unhappy victim upon whom it was practised.

The surgical treatment of gall-stones has of late years made rapid progress. The operation of cholecystotomy, or opening the gall-bladder and removing the stones, which was advised by Sims, has been remarkably successful, particularly in the hands of Lawson Tait. The removal of the gall-bladder, cholecystectomy, has also been practised with success. The indications for operation are: (*a*) Repeated attacks of gall-stone colic, of great severity and danger. (*b*) The presence of a distended gall-bladder, associated with attacks of pain or with fever. Many cases of obstruction of the cystic duct with moderate distention of the gall-bladder produce little or no inconvenience, and perfect recovery may take place with contraction and obliteration. (*c*) When a gall-stone is permanently lodged in the common duct, and presents the group of symptoms above described. It must, however, be borne in mind that, contrary to the experiences of Charcot and other French writers, three of my cases recovered—one after persistence of the condition for eight months, another for three years; two died of the effects of the prolonged jaundice, and two after operation. The question, then, of advising removal in such cases should depend largely upon the personal methods and success of the surgeon who is available. The common duct has been explored and gall-stones removed from it. The operation is necessarily much more serious and difficult than that upon the gall-bladder.

IV. CIRRHOSIS.

Definition.—A chronic disease of the liver, characterized by a gradual destruction of liver-cells and an overgrowth of connective-tissue elements, in consequence of which the organ becomes hard and usually small.

Etiology.—The disease occurs most frequently in middle-aged males. It has been regarded as rare in children, except in the syphilitic form, but Palmer Howard collected 63 cases, to which list Hatfield, in a further search of the literature, has been able to add 93, so that its occurrence in early life is more common than has been supposed.

The following are the recognized factors in inducing the disease: (*a*) *Alcohol.*—The abuse of spirits is the common cause. It is more frequent in countries in which strong spirits are taken than in those in which malt liquors and wines are used. The change results from the irritative effect of the strong solution of alcohol absorbed from the stomach. The fusel oil is thought to be the offending material. Similar effects are doubtless produced by other substances, such as rich, highly seasoned foods, or, as has been suggested, by ptomaines and other alkaloids.

(*b*) *Syphilis.*—We have already considered (under Syphilis) the forms of cirrhosis, diffuse and gummatous, produced by this poison.

(*c*) *Cyanotic Congestion.*—In cases of chronic disease of the heart and lungs the liver is in a condition of persistent venous hyperæmia, in consequence of which the central cells of the liver lobules atrophy and there is hyperplasia of the connective tissue.

(*d*) *Malaria.*—Sclerosis of the liver may follow prolonged malarial poisoning. In this country it is very rare.

(*e*) *Tuberculosis.*—We have already referred to the sclerotic changes in the liver produced by tuberculosis. It rarely, if ever, induces a condition which can be recognized clinically.

(*f*) *Scarlet Fever.*—The fact noted by Klein that in scarlet fever there was an infiltration with small cells, an acute interstitial hepatitis, gives a clew to the occurrence of some of the cases of cirrhosis of the liver in children. In other infectious diseases, too, such as typhoid, there are localized necrotic areas which must be replaced by connective tissue. In the cirrhosis of early life, excluding the alcoholic and syphilitic cases, the acute infectious diseases are probably the important antecedents.

(*g*) *Rickets.*—The enlargement of the liver in this disease is associated with increase in the connective tissue, which surrounds the individual lobules and produces changes in the bile-ducts (Hodgben).

(*h*) *Anthracosis.*—It occasionally happens in coal-miners that the carbon pigment reaches the liver in large quantities, is deposited in the connective tissue about the portal canal, and may lead to a variety of cirrhosis, which has been described by Welch.

In animals, artificial obstruction of the bile-passages results in cirrhosis, but in man there may be persistent stenosis of the common duct or ob-

struction without marked increase in the connective tissue. The causes which induce the cirrhosis which we meet at the bedside are alcohol and syphilis.

Morbid Anatomy.—Practically on the post-mortem table we see cirrhosis in four well-characterized forms:

(*a*) *The Atrophic Cirrhosis of Laennec.*—The organ is greatly reduced in size and may be deformed. The weight is sometimes not more than a pound or a pound and a half. It presents numerous granulations on the surface; is firm, hard, and cuts with great resistance. The substance is seen to be made up of greenish-yellow islands, surrounded by grayish-white connective tissue. This yellow appearance of the liver induced Laennec to give it the name of cirrhosis.

(*b*) *Fatty Cirrhosis.*—Even in the atrophic form the fat is increased, but in typical examples of this variety the organ is not reduced in size, but is enlarged, smooth or very slightly granular, anæmic, yellowish white in color, and resembles an ordinary fatty liver. It is, however, firm, cuts with resistance, and microscopically shows a great increase in the connective tissue. This form is quite as common in this country as the atrophic variety. It occurs most frequently in beer-drinkers.

(*c*) *Hypertrophic Cirrhosis.*—Enlargement of the liver occurs in the early stage of the ordinary atrophic cirrhosis, but the increase is moderate and largely due to hyperæmia. The fatty cirrhotic liver is also large, and may reach a hand's-breadth below the costal margin. The term hypertrophic cirrhosis should be restricted to the form described by French writers, which is also known as *biliary* cirrhosis. Unfortunately, this has been used by some writers to include as well the cases in which there has been permanent occlusion of the duct, either by stricture or a calculus; the induration, however, is slight under these circumstances and hypertrophy very rare. It seems best to limit the terms *biliary* and *hypertrophic* cirrhosis to the form characterized by permanent enlargement of the liver, a marked involvement of the smaller biliary canaliculi, and retention in an unusual degree, in comparison with atrophic cirrhosis, of the number and form of the liver-cells, in spite of the great increase of the lobular connective tissue. In this form the liver is greatly enlarged; in one of my cases it weighed seven pounds. The surface is smooth, it is exceedingly firm, resists cutting, and presents on section a deep greenish-yellow color. All of my cases have been in hard drinkers.

(*d*) *Perihepatitis; Glissonian Cirrhosis.*—In this form the liver is greatly reduced in size, much altered in shape, and everywhere surrounded by a firm grayish-white membrane, sometimes of semi-cartilaginous consistence, varying from 10 to .5 mm. in thickness. This fibrous investment can be stripped off readily, and the liver substance may look almost normal, but usually shows cirrhotic changes. The capsular thickening may be slight, and the portal connective tissue chiefly involved. The capsule of the spleen is, as a rule, similarly affected, and both processes are asso-

ciated with a proliferative peritonitis. The condition is most frequent as a result of alcohol, but occurs also in instances of cyanotic induration.

The two essential elements in cirrhosis are destruction of liver-cells and obstruction to the portal circulation.

In an autopsy on a case of atrophic cirrhosis the peritonæum is usually found to contain a large quantity of fluid, the membrane is opaque, and there is chronic catarrh of the stomach and of the small intestines. The kidneys are sometimes cirrhotic, the bases of the lungs may be much compressed by the ascitic fluid, the heart often shows marked degeneration, and arterio-sclerosis is usually present. A remarkable feature is the association of acute tuberculosis with cirrhosis. In seven cases of my series the patients died with either acute tuberculous peritonitis or acute tuberculous pleurisy. Pitt states that twenty-two and a half per cent of the cases of cirrhosis dying in Guy's Hospital during twelve years had acute tuberculosis.

The compensatory circulation is usually readily demonstrated. It is carried out by the following set of vessels: (1) The accessory portal system of Sappey, of which important branches pass in the round and suspensory ligaments and unite with the epigastric and mammary systems. These vessels are numerous and small. Occasionally a large single vein, which may attain the size of the little finger, passes from the hilus of the liver in the round ligament, and joins the epigastric veins at the navel. Although this has the position of the umbilical vein, it is usually, as Sappey showed, a para-umbilical vein—that is, an enlarged vein by the side of the obliterated umbilical vessel. There may be produced about the navel a large bunch of varices, the so-called caput Medusæ. Other branches of this system occur in the gastro-epiploic omentum, about the gall-bladder, and, most important of all, in the suspensory ligament. These latter form large branches, which anastomose freely with the diaphragmatic veins, and so unite with the vena azygos. (2) By the anastomosis between the œsophageal and gastric veins. The veins at the lower end of the œsophagus may be enormously enlarged, producing varices which project on the mucous membrane. (3) The communications between the hæmorrhoidal and the inferior mesenteric veins. The freedom of communication in this direction is very variable, and in some instances the hæmorrhoidal veins are not much enlarged. (4) The veins of Retzius, which unite the radicles of the portal branches in the intestines and mesentery with the inferior vena cava and its branches. To this system belong the whole group of retroperitoneal veins, which are in most instances enormously enlarged, particularly about the kidneys, and which serve to carry off a considerable proportion of the portal blood.

Symptoms.—(*a*) *Of the Atrophic Form.*—The most extreme grade of atrophic cirrhosis may exist without symptoms. *So long as the compensatory circulation is maintained* the patient may suffer little or no inconvenience. The remarkable efficiency of this collateral circulation is well

seen in those rare instances of permanent obliteration of the portal vein, which may exist for many years.

The symptoms may be divided into two groups—obstructive and toxic.

Obstructive.—The overfilling of the blood-vessels of the stomach and intestine leads to chronic catarrh, and the patients suffer with nausea and vomiting, particularly in the morning; the tongue is furred and the bowels are irregular. Hæmorrhage from the stomach may be an early symptom; it is often profuse and liable to recur. It seldom proves fatal. The amount vomited may be remarkable, as in a case already referred to, in which ten pounds were ejected in seven days. Following the hæmatemesis melæna is common. Enlargement of the spleen occurs from the chronic congestion. The organ can usually be felt. Evidences of the establishment of the collateral circulation are seen in the enlarged epigastric and mammary veins, more rarely in the presence of the caput Medusæ and in the development of hæmorrhoids. The distended venules in the lower thoracic zone along the line of attachment of the diaphragm are not specially marked in cirrhosis. The most striking feature of failure in the compensatory circulation is ascites, the effusion of serous fluid into the peritoneal cavity. The conditions under which this occurs are still obscure. The abdomen gradually distends, may reach a large size, and contain as much as 15 or 20 litres. Œdema of the feet may precede or develop with the ascites. The dropsy rarely becomes general.

Jaundice is usually slight, and was present in only 35 of 130 cases of cirrhosis reported by Fagge. The skin has frequently a sallow, slightly icteroid tint. The urine is often reduced in amount, contains urates in abundance, often a slight amount of albumen, and, if jaundice is intense, tube-casts. The disease may be afebrile throughout, but in many cases, as shown by Carrington, there is slight fever, from 100° to 102·5°.

Examination in the early stage of the disease may show moderate enlargement of the liver, which may be painful on pressure. At this period the patient may come under observation for dyspepsia, hæmatemesis, slight jaundice, or nervous symptoms. Later in the disease, the patient has an unmistakable hepatic facies; he is thin, the eyes are sunken, the conjunctivæ watery, the nose and cheeks show distended venules, and the complexion is muddy or icteroid. On the enlarged abdomen the vessels are distended, and a bunch of dilated veins may surround the navel. When much fluid is in the peritonæum it is impossible to make a satisfactory examination, but after withdrawal the area of liver dulness is found to be diminished, particularly in the middle line, and on deep pressure the edge of the liver can be detected, and occasionally the hard, firm, and even granular surface. The spleen can be felt in the left hypochondriac region. Examination of the anus may reveal the presence of hæmorrhoids.

Toxic Symptoms.—At any stage of atrophic cirrhosis the patient may develop cerebral symptoms, either a noisy, joyous delirium, or stupor, coma, or even convulsions. The condition is not infrequently mistaken for

uræmia. The nature of the toxic agent is not yet settled. The symptoms may develop without jaundice, and cannot be attributed to cholæmia, and they may come on in hospital when the patient has not had alcohol for weeks.

The fatty cirrhotic liver may produce symptoms similar to those of the atrophic form, but it more frequently is latent and is found accidentally in topers who have died from various diseases. The greater number of the cases clinically diagnosed as cirrhosis with enlargement come in this division.

(*b*) *Hypertrophic* or biliary cirrhosis has a definite and distinctive symptomatology. The liver may be enlarged for months or even years. Jaundice persists for some time, on which point French writers lay great stress. It may, however, come on acutely with the other symptoms. It is intense, like an obstructive jaundice, but, as a rule, the stools are bile-stained. It may continue for a long time without the development of other symptoms; then delirium sets in and all the features of an acute febrile jaundice. The tongue is dry, the pulse rapid, the temperature ranges from 102° to 104°, and petechiæ occur on the skin. The patient may present every feature of acute yellow atrophy, including even the convulsive seizures. The attack in one of my cases proved fatal within ten days; in another it was prolonged for three weeks. Ascites does not develop. The enlargement of the liver may be the sole diagnostic criterion between these cases and acute yellow atrophy. I do not know, however, of the occurrence of leucin or tyrosin in the urine in this condition.

(*c*) The perihepatitis with cirrhosis cannot be distinguished from the ordinary atrophic form.

Diagnosis.—With ascites, a well-marked history of alcoholism, the hepatic facies, and hæmorrhage from the stomach or bowels, the diagnosis is rarely doubtful. If, after withdrawal of the fluid, the spleen is found to be enlarged and the liver either not palpable or, if it is enlarged, hard and regular, the probabilities in favor of cirrhosis are very great. In the early stages of the disease, when the liver is increased in size, it may be impossible to say whether it is a cirrhotic or a fatty liver. The differential diagnosis between common and syphilitic cirrhosis can sometimes be made. A marked history of syphilis or the existence of other syphilitic lesions, with great irregularity in the surface or at the edge of the liver, are the points in favor of the latter. Thrombosis or obliteration of the portal vein can rarely be differentiated In the case of fibroid transformation of the portal vein which came under my observation, the collateral circulation had been established for years, and the symptoms were simply those of extreme portal obstruction, such as occur in cirrhosis. Thrombosis of the portal vein is frequent in cirrhosis and may be characterized by a rapidly developing ascites.

Prognosis.—The prognosis is, as a rule, bad. When the collateral circulation is fully established the patient may have no symptoms what-

ever. Three cases of advanced atrophic cirrhosis have died under my observation of other affections without presenting during life any symptoms pointing to disease of the liver. There are instances, too, of enlargement of the liver, slight jaundice, cerebral symptoms, and even hæmatemesis, in which the liver becomes reduced in size, the symptoms disappear, and the patient may live in comparative comfort for many years. There are many cases, too, in which, after one or two tappings, the symptoms have disappeared and the patients have apparently recovered.

Treatment.—Ordinary cirrhosis of the liver is an incurable disease. Many writers, speaking of the curability of certain forms, show a lack of appreciation of the essential conditions upon which the symptoms depend. So far as we have any knowledge, no remedies at our disposal can alter or remove the cicatricial connective tissue which constitutes the *materia peccans* in ordinary cirrhosis. On the other hand, we know that extreme grades of contraction of the liver may persist for years without symptoms when the compensatory circulation exists. The so-called cure of cirrhosis means the re-establishment of this compensation; and it would be as unreasonable to speak of healing a chronic valvular lesion when with digitalis we have restored the circulatory balance as it is to speak of curing cirrhosis of the liver when by tapping and other measures the compensation has in some way been restored.

The patient should abstain entirely from alcohol, and, if possible, should take a milk diet, which has been highly recommended by Semmola. In any case, the diet should be nutritious, but not too rich. Measures should be employed to reduce the gastro-intestinal catarrh, and the patient should lead a quiet, out-of-door life and keep the skin active, the bowels regular, and the urine abundant. In non-syphilitic cases it is useless to give either mercury or iodide of potassium. When a well-marked history of syphilis exists these remedies should be used, but neither of them has any more influence upon the development of a new growth of connective tissue in the liver than it has upon the progressive development of a scar tissue in a keloid or in an ordinary developing cicatrix. The ascites should be tapped early, and the operation may be repeated so soon as the distention becomes distressing. The continuous drainage with a Southey's tube may be employed. It is much better to resort to tapping early if after a few days' trial the fluid does not subside rapidly under the use of saline purges. From half an ounce to an ounce and a half of sulphate of magnesia may be given in as little water as possible half an hour before breakfast. Elaterium, the compound jalap powder, or the bitartrate of potash may also be employed. Digitalis and squills are often useful. In the syphilitic cases or when syphilis is suspected iodide of potassium may be given in doses of from fifteen to thirty drops of the saturated solution three times a day, and mercury, which is conveniently given with squills and digitalis in the form of Addison's or Niemeyer's pill. A case of well-marked syphilitic cirrhosis with recurring ascites, in which tapping was resorted to on eight

or ten occasions, took this pill at intervals for a year with the greatest benefit, and subsequently had four years of tolerably good health.

V. ABSCESS OF THE LIVER.

Etiology.—Suppuration within the liver, either in the parenchyma or in the blood or bile passages, occurs under the following conditions:

(1) The tropical abscess. In hot climates this form may develop idiopathically, but more commonly follows dysentery. It frequently occurs among Europeans in India, particularly those who drink alcohol freely and are exposed to great heat. The relation of this form of abscess to dysentery is still under discussion, and Anglo-Indian practitioners are by no means unanimous on the subject. Certainly cases may develop without a history of previous dysentery, and there have been fatal cases without any affection of the large bowel. In this country the large solitary tropical abscess also occurs, oftenest in the Southern States. In Baltimore it is not very infrequent, as may be judged from the fact that during two years there have been at my clinic five cases, and I know of the occurrence of three or four additional cases during this time in the city.

The relation of this form of abscess to the *amœba coli* has been carefully studied by Kartulis and exhaustively considered in a monograph by Councilman and Lafleur. The descriptions and illustrations of these authors are most convincing as to the direct etiological association of this organism with liver abscess. Clinically the patient may have *amœba coli* in the stools and well-marked signs of liver abscess without marked symptoms of dysentery and even with the fæces well formed.

(2) Traumatism is an occasional cause. The injury is generally in the hepatic region. Two instances have come under my notice of it in brakemen who were injured while coupling cars. Injury of the head is not infrequently followed by liver abscess.

(3) Embolic or pyæmic abscesses are the most numerous, and may develop in a general pyæmia from any cause or follow foci of suppuration in the territory of the portal vessels. The infective agents may reach the liver through the hepatic artery, as in those cases in which the original focus of infection is in the area of the systemic circulation; though it may happen occasionally that the infective agent, instead of passing through the lungs, reaches the liver through the inferior vena cava and the hepatic veins. A remarkable instance of multiple abscesses of arterial origin was afforded by the case of aneurism of the hepatic artery reported by Ross and myself. Infection through the portal vein is much more common. It results from dysentery and other ulcerative affections of the bowels, appendicitis, occasionally after typhoid fever, in rectal affections, and in abscesses in the pelvis. In these cases the abscesses are multiple and, as a rule, within the branches of the portal vein—suppurative pylephlebitis.

(4) A not uncommon cause of suppuration is inflammation of the bile-passages caused by gall-stones, more rarely by parasites—suppurative cholangitis.

In some instances of tuberculosis of the liver the affection is chiefly of the bile-ducts, with the formation of multiple tuberculous abscesses containing a bile-stained pus.

(5) Foreign bodies and parasites. In rare instances foreign bodies, such as a needle, may pass from the stomach or gullet, lodge in the liver, and excite an abscess, or, as in several instances which have been reported, a foreign body, such as a needle or a fish-bone, may perforate a branch or the portal vein itself and induce extensive pylephlebitis. Echinococcus cysts frequently cause suppuration; the penetration of round worms into the liver less commonly; and most rarely of all the liver-fluke.

Morbid Anatomy.—(*a*) *Of the Solitary or Tropical Abscess.*—This is not always single; there may be two or even more large abscess cavities, ranging in size from an orange to a child's head. The largest-sized abscess may contain from three to six litres of pus and involve more than three fourths of the entire organ. In Waring's statistics, sixty-two per cent of the cases were single. The abscess in nearly seventy per cent of the cases was in the right lobe, more toward the convexity than the concave side. In long-standing cases the abscess-wall may be firm and thick, but, as a rule, the cavity possesses no definite limiting membrane, and section of the wall shows an internal layer, grayish in color, shreddy, and made up of necrotic liver substance, pus-cells, and amœbæ; a middle layer, brownish red in color; and an external zone of hyperæmic liver tissue. The pus is often reddish brown in color, closely resembling anchovy sauce. In other instances it is grayish white, mucoid, and may be quite creamy. The odor is at times very peculiar. In one instance it had the sour smell of chyme, though no connection with the stomach was found. In a recent case of amœbic dysentery there were multiple miliary abscesses in the liver, all of which contained amœbæ.

The bacteriological examination of the contents show that as a rule the pus is sterile (Kartulis). The termination of this form of abscess may be as follows, as noted in Waring's 300 cases: Remained intact, fifty-six per cent; opened by operation, sixteen per cent; perforated the right pleura, nearly five per cent; ruptured into the right lung, nine per cent; ruptured into the peritonæum, five per cent; ruptured into the colon, nearly three per cent; and there were in addition instances which ruptured into the hepatic and bile-vessels and into the gall-bladder.

(*b*) *Of Septic and Pyæmic Abscesses.*—These are always multiple, though occasionally, following injury, there may be a large solitary collection of pus.

In suppurative pylephlebitis the liver is uniformly enlarged. The capsule may be smooth and the external surface of the organ of normal appearance. In other instances, numerous yellowish-white points appear

beneath the capsule. On section there are isolated pockets of pus, either having a round outline or in some places distinctly dendritic, and from these the pus may be squeezed. They look like small, solitary abscesses, but, on probing, are found to communicate with the portal vein and to represent its branches, distended and suppurating. The entire portal system within the liver may be involved; sometimes territories are cut off by thrombi. The suppuration may extend into the main branch or even into the mesenteric and gastric veins. The pus may be fetid and is often bile-stained; it may, however, be thick, tenacious, and laudable. In suppurative cholangitis there is usually obstruction by gall-stones, the ducts are greatly distended, the gall-bladder enlarged and full of pus, and the branches within the liver are extremely distended, so that on section there is an appearance not unlike that described in pylephlebitis.

Suppuration about echinococcus cysts may be very extensive, forming enormous abscesses, the characters of which are at once recognized by the remnants of the cysts.

Symptoms.—(*a*) *Of the Large Solitary Abscess.*—In the tropics there are instances in which the abscess appears to be latent and to run a course without definite symptoms, and death may occur suddenly from rupture.

Fever, pain, enlargement of the liver, and the development of a septic condition are the important symptoms of hepatic abscess. The temperature is elevated at the outset and is of an intermittent or septic type. It is irregular, and may remain normal or even subnormal for a few days; then the patient has a rigor and the temperature rises to 103° or higher. Owing to this intermittent character of the fever the cases are usually, in this latitude, mistaken for malaria. The fever may rise every afternoon without a rigor. Profuse sweating is common, particularly when the patient falls asleep. In chronic cases there may be little or no fever. At the time of writing, there is in one of my wards a patient with liver abscess which has perforated the lung who still coughs up pus, but whose temperature has been normal for weeks. The pain is variable, and is usually referred to the back or shoulder; or there is a dull aching sensation in the right hypochondrium. When turned on the left side, the patient often complains of a heavy, dragging sensation, so that he usually prefers to lie on the right side; at least, this has been the case in a majority of the instances which have come under my observation. Pain on pressure over the liver is usually present, particularly deep pressure at the costal margin in the nipple line.

The enlargement of the liver is most marked in the right lobe, and, as the abscess cavity is usually situated more toward the upper than the under surface, the increase in volume is upward and to the right, not downward, as in cancer and the other affections producing enlargement. Percussion in the mid-sternal and parasternal lines may show a normal limit. At the nipple-line the curve of liver dulness begins to rise, and in the mid-

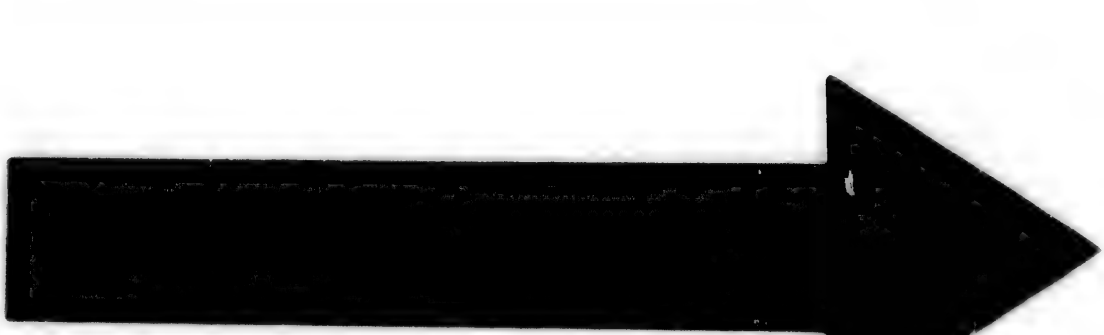
axillary it may reach the fifth rib, while behind, near the spine, the area of dulness may be almost on a level with the angle of the scapula. Of course there are instances in which this characteristic feature is not present, as when the abscess occupies the left lobe. The enlargement of the liver may be so great as to cause bulging of the right side, and the edge may project a hand's-breadth or more below the costal margin. In such instances the surface is smooth. Palpation is painful, and there may be fremitus on deep inspiration. In some instances fluctuation may be detected. Adhesions may form to the abdominal wall and the abscess may point below the margin of the ribs, or even in the epigastric region. In many cases the appearance of the patient is suggestive. The skin has a sallow, slightly icteroid tint, the face is pale, the complexion muddy, the conjunctivæ are infiltrated, and often slightly bile-tinged. There is in the facies and in the general appearance of the patient a strong suggestion of the existence of abscess. There is no internal affection associated with suppuration which gives, I think, just the same hue as certain instances of abscess of the liver. Marked jaundice is rare. Diarrhœa may be present and may give an important clew to the nature of the case, particularly if amœbæ are found in the stools. Constipation may occur.

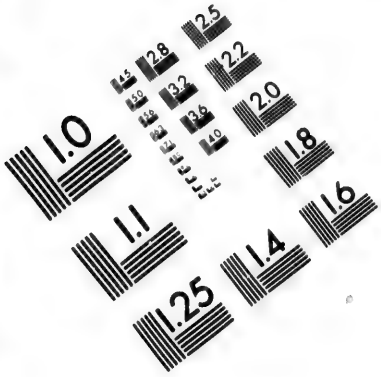
Remarkable and characteristic symptoms arise when the abscess invades the lung. The extension may occur through the diaphragm, without actual rupture, and with the production of a purulent pleurisy and invasion of the lung. In four cases of this kind, which have been under observation recently, the patients gradually developed a severe cough, usually of an aggravated and convulsive character, there were signs of involvement at the base of the right lung, defective resonance, feeble tubular breathing, and increase in the tactile fremitus; but the most characteristic feature was the presence of a reddish-brown expectoration of a brick-dust color, resembling anchovy sauce. This, which was noted originally by Budd, was present in our cases, and in addition Reese and Lafleur found in all *amœbæ coli* identical with those which exist in the liver abscess and in the stools. They are present in variable numbers and display active amœbic movements. The brownish tint of the expectoration is due to blood-pigment and blood-corpuscles, and there may be orange-red crystals of hæmatoidin.

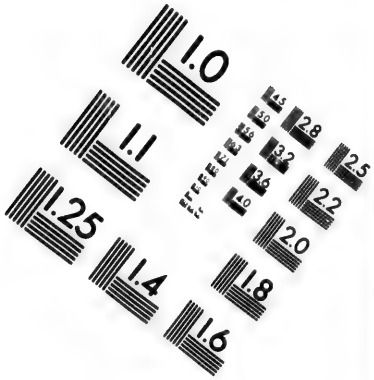
The abscess may perforate externally, as mentioned already, or into the stomach or bowel; occasionally into the pericardium. The duration of this form is very variable. It may run its course and prove fatal in six or eight weeks or may persist for several years.

The prognosis is serious, as the mortality is more than fifty per cent. The death-rate has been lowered of late years, owing to the greater fearlessness with which surgeons now attack these cases.

(*b*) *Of the Pyæmic Abscess and Suppurative Pylephlebitis.*—Clinically these conditions cannot be separated. Occurring in a general pyæmia, no special features may be added to the case. When there is suppuration







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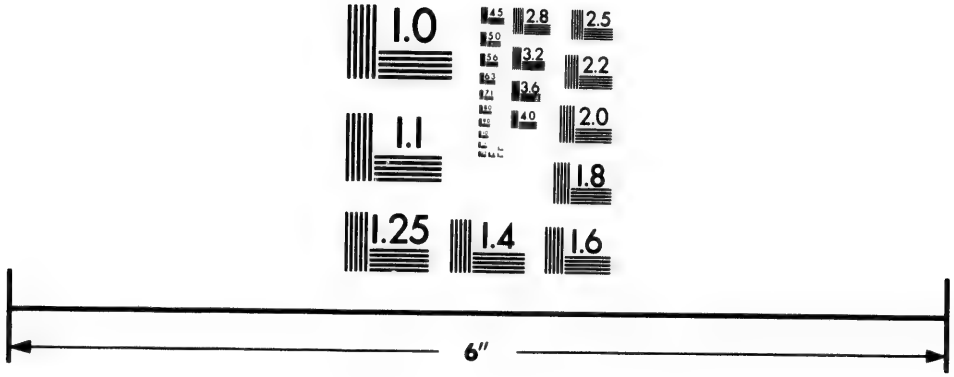

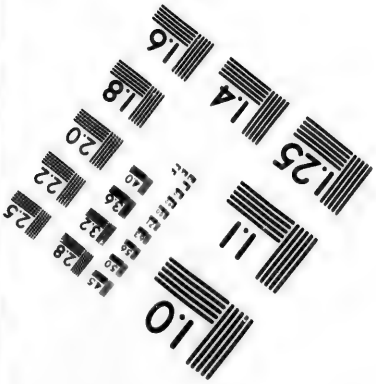

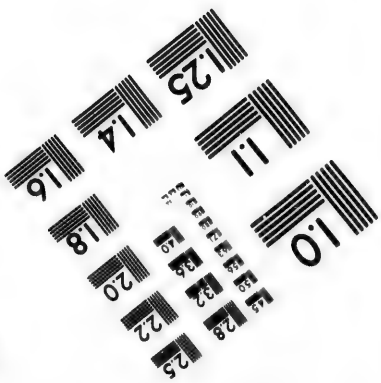

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within the portal vein the liver is uniformly enlarged and tender, though pain may not be a marked feature. There is an irregular, septic fever, and the complexion is muddy, sometimes distinctly icteroid. The features are indeed those of pyæmia, plus a slight icteroid tinge, and an enlarged and painful liver. The latter features alone are peculiar. The sweats, chills, prostration, and fever have nothing distinctive.

Diagnosis.—Abscess of the liver may be confounded with intermittent fever, a common mistake in malarial regions. Practically an intermittent fever which resists quinine is not malarial. Laveran's organisms are also absent from the blood. When the abscess bursts into the pleura a right-sided empyema is produced and perforation of the lung usually follows. When the liver abscess has been latent and dysenteric symptoms not marked, the condition may be considered empyema or abscess of the lung. In such cases the anchovy-sauce-like color of the pus and the presence of the amœbæ will enable one to make a definite diagnosis, as has been done in cases by Lafleur. Perforation externally is readily recognized, and yet in an abscess cavity in the epigastric region it may be difficult to say whether it has proceeded from the liver or is in the abdominal wall. When the abscess is large, and the adhesions are so firm that the liver does not descend during inspiration, the exploratory needle does not make an up-and-down movement during aspiration. In an instance of this kind which I saw with Hearn at the Philadelphia Hospital, all the features, local and general, seemed to point to abscess in the abdominal wall, but the operation revealed a large perforating abscess cavity in the left lobe of the liver. The diagnosis of suppurating echinococcus cyst is rarely possible, except in Australia and Iceland, where hydatids are so common. In the only case which has come under my observation, the innumerable tumors scattered throughout the abdomen and the great size of the liver led, not unnaturally, in spite of the occurrence of septic symptoms, to the diagnosis of cancer.

Perhaps the most important affection from which suppuration within the liver is to be separated is the intermittent hepatic fever associated with gall-stones. Of the cases reported a majority have been considered due to suppuration, and in two of my cases the liver had been repeatedly aspirated. Post-mortem examinations have shown conclusively that the high fever and chills may recur at intervals for years without suppuration in the ducts. The distinctive features of this condition are paroxysms of fever with rigors and sweats—which may occur with great regularity, but which more often are separated by long intervals—the deepening of the jaundice after the paroxysms, the entire apyrexia in the intervals, and the maintenance of the general nutrition. The time element also is important, as in some of these cases the disease has lasted for several years. Finally, it is to be remembered that abscess of the liver, in temperate climates at least, is invariably secondary, and the primary source must be carefully sought for, either in dysentery, slight ulceration of the rectum, suppurating

hæmorrhoids, ulcer of the stomach, or in suppurative diseases of other parts of the body, particularly in the skull or in the bones.

In suspected cases, whether the liver is enlarged or not, exploratory aspiration may be performed without risk. The needle may be entered in the anterior axillary line in the lowest interspace, or in the seventh interspace in the mid-axillary line, or over the centre of the area of dulness behind. The patient should be placed under ether, for it may be necessary to make several deep punctures. It is not well to use too small an aspirator. No ill effects follow this procedure, even though blood may leak into the peritoneal cavity. Extensive suppuration may exist, and yet be missed in the aspiration, particularly when the branches of the portal vein are distended with pus.

Treatment.—Pyæmic abscesses and suppurative pylephlebitis are invariably fatal. Surgical measures are not justified in these cases, unless an abscess shows signs of pointing As the abscesses associated with dysentery are often single, they afford a reasonable hope for operation. If, however, the patient is expectorating the pus, if the general condition is good and the hectic fever not marked, it is best to defer operation, as many of these instances recover spontaneously. The large single abscesses offer the best chance for operation.

The general medical treatment of the cases is that of ordinary septicæmia.*

VI. NEW GROWTHS IN THE LIVER.

These may be cancer, either primary or secondary, sarcoma, or angioma.

Etiology.—Cancer of the liver is third in order of frequency of internal cancer. It is rarely primary, usually secondary to cancer in other organs. It is a disease of late adult life. According to Leichtenstern, over fifty per cent of the cases occur between the fortieth and the sixtieth years. It occasionally occurs in children. Women are attacked less frequently than men. It is stated by some authors that secondary cancer is more common in women, owing to the frequency of cancer of the uterus. Heredity is believed to have an influence in from fifteen to twenty per cent.

In many cases trauma is an antecedent, and cancer of the bile-passages is associated in many cases with gall-stones. Cancer is stated to be less common in the tropics. Its relative proportion to other diseases may be judged from the fact that among the first three thousand patients admitted to the wards of the Johns Hopkins Hospital there were seven cases of cancer of the liver.

* For general rules and the modern surgical treatment of the condition, the reader is referred to Godlee's lectures, British Medical Journal, vol. i, 1890.

Morbid Anatomy.—The following forms of new growths occur in the liver and have a clinical importance:

Cancer.—(1) *Primary cancer*, of which three forms may be recognized.*

(*a*) The *massive cancer*, which causes great enlargement and on section shows a uniform mass of new growth, which occupies a large portion of the organ. It is grayish white, usually not softened, and is abruptly outlined from the contiguous liver substance.

(*b*) *Nodular cancer*, in which the liver is occupied by nodular masses, some large, some small, irregularly scattered throughout the organ. Usually in one region there is a larger, perhaps firmer, older-looking mass, which indicates the primary seat, and the numerous nodules are secondary to it. This form is much like the secondary cancerous involvement, except that it seldom reaches a large size.

(*c*) The third is the remarkable and rare variety, *cancer with cirrhosis*, which forms an anatomical picture perfectly unique and at first very puzzling. The liver is not much enlarged, rarely weighing more than two and a half or three kilogrammes. The surface is grayish yellow, studded over with nodular yellowish masses, resembling the projections in an ordinary cirrhotic liver. On section the cancerous nodules are seen scattered throughout the entire organ, varying in diameter from three to ten or more millimetres and surrounded with fibrous tissue.

Histologically, the primary cancers are epitheliomata—alveolar and trabecular. The character of the cells varies greatly. Some varieties are polymorphous; others small polyhedral; and others again contain giant cells. In rare instances, as in one described by Greenfield, the cells are cylindrical. The trabecular form of epithelioma is also known as adenoma or adeno-carcinoma.

(2) *Secondary Cancer.*—The organ is usually enormously enlarged, and may weigh twenty pounds or more. The cancerous nodules project beneath the capsule, and can be felt during life or even seen through the thin abdominal walls. They are usually disseminated equally, though in rare instances they may be confined to one lobe. The consistence of the nodules varies; in some cases they are firm and hard and those on the surface show a distinct umbilication, due to the shrinking of the fibrous tissue in the centre. These superficial cancerous masses are still sometimes spoken of as "Farre's tubercles." More frequently the masses are on section grayish white in color, or hæmorrhagic. Rupture of blood-vessels is not uncommon in these cases. In one specimen there was an enormous clot beneath the capsule of the liver, together with hæmorrhage into the gall-bladder and into the peritonæum. The secondary cancer shows the same structure as the initial lesion, and is usually either an alveolar or cylindrical carcinoma. Degeneration is common in these second-

* Hanot and Gilbert, Études sur les Maladies du Foie, Paris, 1888.

ary growths; thus the hyaline transformation may convert large areas into a dense, dry, grayish-yellow mass. Extensive areas of fatty degeneration may occur, sclerosis is not uncommon, and hæmorrhages are frequent. Suppuration sometimes follows.

(3) *Cancer of the Bile-Passages.*—Much attention has been given to this of late, and both Zenker and Musser have recently published exhaustive papers on the subject. In 100 cases collected by Musser the large proportion (3 to 1) were in females. Jaundice was present in sixty-nine per cent, and in about the same percentage there was a tumor in the region of the gall-bladder. Courvoisier has collected 100 cases, of which 83 were in men and 17 in women. The association of cancer of the bile-passages with calculi has long been recognized, and they are present in at least seven eighths of all cases. The fundus of the gall-bladder is usually involved first. The process may extend to the common or hepatic ducts, and invasion of the contiguous structures is common. The ducts may be affected primarily.

Sarcoma.—Of primary sarcoma of the liver very few cases have been reported. Secondary sarcoma is more frequent, and many examples of lympho-sarcoma and myxo-sarcoma are on record, less frequently glio-sarcoma or the smooth or striped myoma.

The most important form is the melano-sarcoma, which develops in the liver secondarily to sarcoma of the eye or of the skin. Very rarely melano-sarcoma develops primarily in the liver. Of the reported cases Hanot excludes all but one. In this form the liver is greatly enlarged, is either uniformly infiltrated with the cancer, which gives the cut surface the appearance of dark granite, or there are large nodular masses of a deep black or marbled color. There are usually extensive metastases, and in some instances every organ of the body is involved. Nodules of melano-sarcoma of the skin may give a clew to the diagnosis.

Other Forms of Liver Tumor.—One of the commonest tumors in the liver is the angioma, which occurs as a small, reddish body the size of a walnut, and consists simply of a series of dilated vessels. Occasionally in children angiomata have developed and produced large tumors.

Cysts are occasionally found in the liver, either single, which are not very uncommon, or multiple, when they usually coexist with congenital cystic kidneys.

Symptoms.—It is often impossible to differentiate primary and secondary cancer of the liver unless the primary seat of the disease is evident, as in the case of scirrhus of the breast, or cancer of the rectum, or of a tumor in the stomach, which can be felt. As a rule, cancer of the liver is associated with progressive enlargement; but there are cases of primary nodular cancer, and in the cancer with cirrhosis the organ may not be enlarged. Gastric disturbance, loss of appetite, nausea, and vomiting are frequent. Progressive loss of flesh and strength may be the first symptoms. Pain or a sensation of uneasiness in the right hypochondriac region

may be present, but enormous enlargement of the liver may occur without the slightest pain. Jaundice, which is present in at least one half of the cases, is usually of moderate extent, unless the common duct is occluded. Ascites is rare, except in the form of cancer with cirrhosis, in which the clinical picture is that of the atrophic form. Pressure by nodules on the portal vein or extension of the cancer to the peritonæum may also induce ascites.

Inspection shows the abdomen to be distended, particularly in the upper zone. In late stages of the disease, when emaciation is marked, the cancerous nodules can be plainly seen beneath the skin, and in rare instances even the umbilications. The superficial veins are enlarged. On palpation the liver is felt, a hand's-breadth or more below the costal margin, descending with each inspiration. The surface is usually irregular, and may present large masses or smaller nodular bodies, either rounded or with central depressions. In instances of diffuse infiltration the liver may be greatly enlarged and present a perfectly smooth surface. The growth is progressive, and the edge of the liver may ultimately extend below the level of the navel. Although generally uniform and producing enlargement of the whole organ, occasionally, when the tumor develops from the left lobe, it may form a solid mass, which occupies the epigastric region. By percussion the outline can be accurately limited and the progressive growth of tumor estimated. The spleen is rarely enlarged. Pyrexia is present in many cases, usually a continuous fever, ranging from 100° to 102°; it may be intermittent with rigors. This may be associated with the cancer alone, or, as in one of my cases, with suppuration. Œdema of the feet, from anæmia, usually supervenes. Cancer of the liver kills in from three to fifteen months.

Diagnosis.—The diagnosis is easy when the liver is greatly enlarged and the surface nodular. The smoother forms of diffuse carcinoma may at first be mistaken for fatty or amyloid liver, but the presence of jaundice, the rapid enlargement, and the more marked cachexia will usually suffice to differentiate it. Perhaps the most puzzling conditions occur in the rare cases of enlarged amyloid liver with irregular gummata. The large echinococcus liver may present a striking similarity to carcinoma, but the projecting nodules are usually softer, the disease lasts much longer, and the cachexia is not marked.

Hypertrophic cirrhosis may at first be mistaken for carcinoma, as the jaundice is usually deep and the liver very large; but the absence of a marked cachexia and wasting, and the painless, smooth character of the enlargement are points against cancer. When in doubt in these cases, aspiration may be safely performed, and positive indication may be gained from the materials so obtained. In large, rapidly growing secondary cancers the superficial rounded masses may almost fluctuate and these soft tumor-like projections may contain blood. The form of cancer with cirrhosis can scarcely be separated from atrophic cirrhosis itself. Perhaps

the wasting is more extreme and more rapid, but the jaundice and the ascites are identical. Melano-sarcoma causes great enlargement of the organ. There are frequently symptoms of involvement of other viscera, as the lungs, kidneys, or spleen. Secondary tumors may develop on the skin. A very important symptom, not present in all cases, is melanuria, the passage of a very dark-colored urine, which may, however, when first voided, be quite normal in color. The existence of a melano-sarcoma of the eye, or the history of blindness in one eye, with subsequent extirpation, may indicate at once the true nature of the hepatic enlargement. The secondary tumors may develop some time after the extirpation of the eye, as in a case under the care of J. C. Wilson, at the Philadelphia Hospital, or, as in a case under Tyson at the same institution, the patient may have a sarcoma of the choroid which had never caused any symptoms. Primary cancer of the gall-bladder can rarely be diagnosed. It may be greatly dilated and readily palpable. Occasionally tumors of the kidney or a tumor of the transverse colon may be confounded with it.

The *treatment* must be entirely symptomatic—allaying the pain, relieving the gastric disturbance, and meeting other symptoms as they arise.

VII. FATTY LIVER.

Two different forms of this condition are recognized—the fatty infiltration and fatty degeneration.

Fatty infiltration occurs, to a certain extent, in normal livers, since the cells always contain minute globules of oil.

In fatty degeneration, which is a much less common condition, the protoplasm of the liver-cells is destroyed and the fat takes its place, as seen in cases of malignant jaundice and in phosphorus poisoning.

Fatty liver occurs under the following conditions: (*a*) In association with general obesity, in which case the liver appears to be one of the store-houses of the excessive fat. (*b*) In conditions in which the oxidation processes are interfered with, as in cachexia, profound anæmia, and in phthisis. The fatty infiltration of the liver in heavy drinkers is to be attributed to the excessive demand made by the alcohol upon the oxygen. (*c*) Certain poisons, of which phosphorus is the most characteristic, produce an intense fatty degeneration with necrosis of the liver-cells. The poison of acute yellow atrophy, whatever its nature, acts in the same way.

The fatty liver is uniformly increased in size. The edge may reach below the level of the navel. It is smooth, looks pale and bloodless; on section it is dry, and renders the surface of the knife greasy. The organ may weigh many pounds, and yet the specific gravity is so low that the entire organ floats in water.

The symptoms of fatty liver are not definite. Jaundice is never present; the stools may be light-colored, but even in the most advanced grades

the bile is still formed. Signs of portal obstruction are rare. Hæmorrhoids are not very infrequent. Altogether, the symptoms are ill-defined, and chiefly those of the disease with which the degeneration is associated. In cases of great obesity, the physical examination is uncertain; but in phthisis and cachectic conditions, the organ can be felt, greatly enlarged, smooth, and painless. Fatty livers are among the largest met with at the bedside.

VIII. AMYLOID LIVER.

The waxy, lardaceous, or amyloid liver occurs as part of a general degeneration, associated with cachexias, particularly when the result of long-standing suppuration.

In practice, it is found oftenest in the prolonged suppuration of tuberculous disease, either of the lungs or of the bones. Next in order of frequency are the cases associated with syphilis. Here there may be ulceration of the rectum, with which it is often connected, or chronic disease of the bone, or it may be present when there are no suppurative changes. It is found occasionally in rickets, in prolonged convalescence from the infectious fevers, and in the cachexia of cancer.

The amyloid organ is large, and may attain dimensions equalled only by that of the cancerous organ. Wilks speaks of a liver weighing fourteen pounds. It is solid, firm, resistant, on section anæmic, and has a semitranslucent, infiltrated appearance. Stained with a dilute solution of iodine, the areas infiltrated with the amyloid matter assume a rich mahogany-brown color. The precise nature of this change is still in question. It first attacks the capillaries, usually of the median zone of the lobules, and subsequently the interlobular vessels and the connective tissue. The cells are but little if at all affected.

There are no characteristic *symptoms* of this condition. Jaundice does not occur; the stools may be light-colored, but the secretion of bile persists. The physical examination shows the organ to be uniformly enlarged and painless, the surface smooth, the edges rounded, and the consistence greatly increased. Sometimes the edge, even in very great enlargement, is sharp and hard. The spleen also may be involved, but there are no evidences of portal obstruction.

The *diagnosis* of the condition is, as a rule, easy. Progressive and great enlargement in connection with suppuration of long standing or with syphilis, is almost always of this nature. In rare instances, however, the amyloid liver is reduced in size.

In *leukæmia* the liver may attain considerable size and be smooth and uniform, resembling, on physical examination, the fatty organ. The blood condition at once indicates the true nature of the case.

IX. DISEASES OF THE PANCREAS.

I. HÆMORRHAGE.

Of late years much attention has been paid to this condition, which may prove rapidly fatal and has important medico-legal bearings. F. W. Draper * has reported five cases, in all of which death occurred either suddenly or after a very short illness. The symptoms are thus briefly summarized by Prince:

"The patient, who has previously been perfectly well, is suddenly taken with the illness which terminates his life. . . . When the hæmorrhage occurs the patient may be quietly resting or pursuing his usual occupation. The pain which ushers in the attack is usually very severe, and located in the upper part of the abdomen. It steadily increases in severity, is sharp or perhaps colicky in character. It is almost from the first accompanied by nausea and vomiting; the latter becomes frequent and obstinate, but gives no relief. The patient soon becomes anxious, restless, and depressed; he tosses about, and only with difficulty can be restrained in bed. The surface is cold, and the forehead is covered with a cold sweat. The pulse is weak, rapid, and sooner or later imperceptible. The abdomen becomes tender, the tenderness being located in the upper part of the abdomen or epigastrium. Tympanites is sometimes marked. The temperature in most cases is either normal or below normal. The bowels are apt to be constipated. These symptoms continue without relief; those which are most striking being the pain, vomiting, anxiousness, restlessness, and the state of collapse into which the patient soon falls."

Post mortem, the pancreas is found uniformly infiltrated with blood. Death, as Zenker suggests, is probably due to shock through the solar plexus.

There are cases in which extensive hæmorrhage occurs into the mesentery, retroperitonæum, or mesocolon. In a patient of Bruen's, at the Philadelphia Hospital, who had for some days obscure abdominal symptoms, I found the entire mesentery and retroperitonæum infiltrated with blood-clots. There was no disease of the aorta or of the cœliac branches or of the mesenteric vessels. Isambard Owen has reported a case of sudden death in a woman aged sixty-seven from hæmorrhage into the transverse mesocolon.

* Transactions of the Association of American Physicians, vol. i.

II. ACUTE PANCREATITIS.

(*a*) **Acute Hæmorrhagic Pancreatitis.**—The admirable studies of Fitz* have crystallized our knowledge on this subject, and brought the affection within the scope of the diagnostician. A majority of the cases occur in persons over thirty. Many of the patients had been addicted to alcohol, and many had suffered from attacks of indigestion, occasionally with severe pains and vomiting.

Morbid Anatomy.—The pancreas is found enlarged, and the interlobular tissue infiltrated with blood, and perhaps with clots. In some instances the contiguous tissues may also be hæmorrhagic, and the whole may form a large, firm mass, situated at the upper and back part of the abdominal cavity. The root of the mesentery, the mesocolon, and the omentum may also show hæmorrhages; the other organs may be practically normal. In some instances there can be seen about the lobules areas of opaque white tissue, and upon the omentum and mesentery similar opaque, white specks, which will be referred to subsequently as the fatty necrosis of Balser. In spots the gland-cells may also be found necrotic, while there may be cases showing a marked increase in the fibrous tissue.

The *symptoms* of this condition are remarkable. The attack sets in with violent pain in the abdomen, usually in the upper zone, but in some instances it is general. Nausea and vomiting are present, and usually constipation. Tympanitic distention of the abdomen is of frequent occurrence. Fever may be present, but is an inconstant symptom. There may be early d[illegible]a. Collapse symptoms supervene, and death occurs usually from the s[illegible]to the fourth day, or even earlier. The swelling and infiltration in the region of the pancreas necessarily involve the cœliac plexus, and the stretching of the nerves may account for the agonizing pain and the sudden collapse. In a case which I have reported the semilunar ganglia were swollen, the nerve-cells indistinct, and there was an interstitial infiltration of round cells. The Pacinian corpuscles in the neighborhood of the pancreas were enormously swollen and œdematous.

A *diagnosis* of intestinal obstruction or of acute perforative peritonitis is usually made. A correct diagnosis was made in one case by Fitz, and the possibility of the presence of this condition must be considered in all abdominal cases which come on suddenly with intense pain in the epigastric region, vomiting, and distention of the abdomen. Perforation of a peptic ulcer or perforation from gall-stones might produce similar symptoms, but the previous history would give important indications. In the case in which the diagnosis was made by Fitz, the patient was suddenly seized with severe pain in the epigastrium, followed by vomiting and prostration. The abdomen was distended, temperature slightly elevated, and the bowels were constipated. The diagnosis lay between ob-

* Middleton-Goldsmith Lecture. New York Medical Record, vol. i, 1889.

struction, perforative peritonitis, and acute pancreatitis. Laparotomy was performed, but no obstruction found. The autopsy showed acute hæmorrhagic pancreatitis.

The cases are stated to be uniformly fatal, but recovery may occur, as shown by a case which was admitted to the Johns Hopkins Hospital. Symptoms of obstruction of the bowels had persisted for three or four days, the abdomen was distended, tender, and very painful. I saw the patient on admission, concurred in the diagnosis of probable obstruction, and, as the condition was serious, ordered him to be transferred at once to the operating-room. The coils were distended and injected, and the peritoneal cavity contained a small amount of bloody serum. No obstruction was found, but in the region of the pancreas and at the root of the mesentery there was a dense, thick, indurated mass and there were areas of fat-necrosis in both mesentery and omentum. The patient recovered.

The literature of the past few years shows that this affection is much more frequent than has been supposed. It has a very important clinical and medico-legal bearing.

A point of interest is the relation of the *fat-necrosis* to pancreatic disease. The areas are found in the interlobular pancreatic tissue, in the mesentery, in the omentum, and in the abdominal fatty tissue generally. In the pancreas the lobules are seen to be separated by a dead-white necrotic tissue, which gives a remarkable appearance to the section. In the abdominal fat the areas are usually not larger than a pin's head; they at once attract attention, and may be mistaken, on superficial examination, for miliary tubercles or neoplasms. They may be larger; instances have been reported in which they were the size of a hen's egg. On section they have a soft, tallowy consistence. Langerhans has shown that this substance is a combination of lime with certain fatty acids. They may be crusted with lime, and in a man, aged eighty, who died of Bright's disease, I found the lobules of the pancreas entirely isolated by areas of fatty necrosis with extensive deposition of lime salts. There is no necessary etiological relation between disease of the pancreas and disseminated fatty necrosis of the abdomen. Cases have been found accidentally in laparotomy for ovarian tumor and in instances in which the pancreas has been normal. They may be found in thin persons. The *bacterium coli commune* was present in two cases, with diphtheritic colitis, examined by Welch.

(*b*) **Suppurative Pancreatitis.**—Of twenty-two cases analyzed by Fitz, the majority occurred in adults under forty years of age; seventeen were males. Anatomically, there may be a diffuse suppuration throughout the organ, which is studded with small abscesses. In other instances the abscess cavity is large and the pancreas is converted into an irregular cyst filled with creamy pus. In more chronic cases the abscess may be circumscribed and the contents cheesy. Communications sometimes occur with the duodenum, or the abscess may burst into the peritonæum. Although the disease is usually chronic, it begins with epigastric pain,

vomiting, and sometimes prostration. There is irregular fever, and death may occur in three or four weeks. In more chronic cases there is very slight fever or only occasional paroxysms. The disease may persist for weeks, months, or even for a year.

The symptoms are indefinite and the condition could scarcely be made out during life. Tenderness exists in the epigastrium, or may at times extend to the left and be quite sharply localized over the position of the pancreas, but a circumscribed tumor is rare. Fat-necrosis is not often found post mortem in these cases.

(*c*) **Gangrenous Pancreatitis.**—Fitz has collected fifteen cases. The pancreas may be converted into a dark, slate-colored, stinking mass, or it may lie nearly free in the omental cavity, attached only by a few shreds of fibrous tissue. Complete sequestration of the organ is not uncommon. It may be discharged as a slough from the bowels, and in two cases in which this happened recovery took place. As a rule, acute peritonitis follows. Hæmorrhagic pancreatitis may precede or be associated with it. Death occurs with symptoms of collapse, commonly in from ten to twenty days. Disseminated fat-necrosis is usually present.

III. CHRONIC PANCREATITIS.

The organ is firmer than normal, the interstitial connective tissue is increased, and there is more or less change in the secreting structures. A special interest has been aroused lately in this affection, as it has been frequently found in diabetes. There may be marked pigmentary changes; a similar condition has been found in the liver. Degeneration of the glandular elements is present in these cases. The sclerosis may be associated with calculi in the ducts.

IV. PANCREATIC CYSTS.

These commonly result from the impaction of calculi; either biliary, lodging at the orifice of the common duct, or pancreatic, within the duct of Wirsung. The pancreatic concretions consist usually of carbonate of lime. George Johnston has collected 35 cases from the literature. Obliteration of the duct may also result from cicatricial contraction and occasionally from displacement. Eighteen cases of cysts of the pancreas have been collected by Senn. The chief symptoms are tumor in the epigastric region, usually median, or sometimes to one side. When large it has occupied the whole abdominal cavity, and in such instances the diagnosis of ovarian tumor has usually been made. The tumor may develop rapidly, or may be chronic and last for many years. In some instances the tumor attained a large size within a few weeks. Pain is not neces

sarily present. Fatty diarrhœa did not exist in any of the cases. The stools may be clay-colored, copious, and putrescent.

The diagnosis of the condition must be extremely difficult, yet it seems to have been made in 7 of the 18 cases. Aspiration should be made to determine the nature of the fluid. This has varied considerably, but most frequently has been brownish or chocolate-colored. In only 6 of the 17 cases in which the nature is mentioned was the fluid of a clear serous character.

V. CANCER.

This is usually scirrhus, and may be primary or secondary. It is not common, as may be judged by the analysis by Segre, who found in 11,492 autopsies only 132 tumors of the pancreas, 127 of which were carcinomata, 2 sarcomata, 2 cysts, and 1 syphiloma. In only 12 of the cases of carcinoma was the disease limited to the gland. The head is commonly affected, and the disease may be limited to this part or extend to it from the stomach or intestines.

The symptoms are variable, and a diagnosis is not often possible. There may be stearrhœa, though it is to be remembered that fatty diarrhœa is not invariably associated with disease of the pancreas. Clay-colored, greasy, and loose stools may be present, with undigested food, as noted by T. J. Walker as a symptom of obstruction of the pancreatic duct. Diabetes may coexist. Although the head of the pancreas can be felt in very thin persons, the tumor masses can rarely be palpated. In the analysis of 137 cases by Da Costa, in only 13 was the tumor recognized by palpation. The general symptoms are those of internal carcinoma. Progressive emaciation, loss of strength, and dyspepsia are present. There is pain in the epigastrium, sometimes paroxysmal. When the head of the pancreas is involved jaundice is almost invariably present.

The disease can scarcely ever be distinguished from cancer in the pyloric zone with involvement of the glands in the hilus of the liver. The movable character of the pyloric tumor and the absence of the hydrochloric acid in the vomit are valuable points. Tumor of the transverse colon is more superficial and movable, is often associated with temporary obstruction, and there may be hæmorrhage from the bowels. In a case with progressive emaciation, epigastric pain, and deep-seated, immobile tumor, with the presence of fatty and greasy stools and the gradual development of jaundice, the diagnosis of cancer of the pancreas is probable.

As the wasting proceeds the aortic pulsation is transmitted with great force through the pancreas and transverse colon, and when a tumor is present the diagnosis of aneurism may be made; but in the latter the sac has not an up-and-down jerking pulsation, but is distensile. In doubtful tumors in this region the examination should also be made in the knee-elbow position.

Of other new growths in the pancreas, tubercle may be mentioned as a rare occurrence; a few cases of syphiloma have been described.

The treatment of new growths in the pancreas is entirely symptomatic.

X. DISEASES OF THE PERITONÆUM.

I. ACUTE GENERAL PERITONITIS.

Definition.—Acute inflammation of the peritonæum.

Etiology.—The condition may be primary or secondary.

(*a*) **Primary, Idiopathic Peritonitis.**—Considering how frequently the pleura and pericardium are primarily inflamed the rarity of idiopathic inflammation of the peritonæum is somewhat remarkable. It may follow cold or exposure and is then known as rheumatic peritonitis. No instance of the kind has come under my notice. Occasionally in Bright's disease acute peritonitis develops as a terminal event.

(*b*) **Secondary Peritonitis** is due to extension of inflammation from, or perforation of one of the organs covered by the peritonæum. Peritonitis from extension may follow inflammation of the stomach or intestines, extensive ulceration in these parts, cancer, acute suppurative inflammations of the spleen, liver, pancreas, retroperitoneal tissues, and the pelvic viscera.

Perforative peritonitis is the most common, following external wounds, perforation of ulcer of the stomach or bowels, perforation of the gall-bladder, abscess of the liver, spleen, or kidneys. Two important causes are appendicitis and suppurating inflammation about the Fallopian tubes and ovaries. There are instances in which peritonitis has followed rupture of an apparently normal Graafian follicle.

The peritonitis of septicæmia and pyæmia is almost invariably the result of a local process. An exceedingly acute form of peritonitis may be caused by the development of tubercles on the membrane.

Morbid Anatomy.—In recent cases, on opening the abdomen the intestinal coils are distended and glued together by lymph, and the peritonæum presents a patchy, sometimes a uniform injection. The exudation may be: (*a*) Fibrinous, with little or no fluid, except a few pockets of clear serum between the coils. (*b*) Sero-fibrinous. The coils are covered with lymph, and there is in addition a large amount of a yellowish, sero-fibrinous fluid. In instances in which the stomach or intestine is perforated this may be mixed with food or fæces. (*c*) Purulent, in which the exudate is either thin and greenish yellow in color, or opaque white and creamy. (*d*) Putrid. Occasionally in puerperal and perforative peritonitis, particularly when the latter has been caused by cancer, the exudate is thin, grayish green in color, and has a gangrenous odor. (*e*) Hæmorrhagic. This is sometimes found as an admixture in cases of acute peri-

tonitis following wounds, and occurs in the cancerous and tuberculous forms.

The amount of the effusion varies from half a litre to twenty or thirty litres. There are probably essential differences between the various kinds of peritonitis, and bacteriology is beginning to give us valuable information on this point. Of the species of micro-organisms which have been found in peritoneal exudates, the pyogenic micrococci and the *bacterium coli commune* are the most common, sometimes one species, often several species being found in the same case. The *streptococcus pyogenes* is by far the most frequent cause of puerperal peritonitis. This species, and still oftener the *staphylococcus pyogenes aureus*, or *albus*, are found in peritonitis consecutive to laparotomy. The *bacterium coli commune*, usually combined with other bacteria, is met with especially in peritonitis secondary to intestinal perforation. The *diplococcus pneumoniæ* has been found several times in peritoneal exudates. The *amœba coli* occurred in numbers in the thin fibrinous effusion in one of our cases of amœbic dysentery.

Symptoms.—In the perforative and septic cases the onset is marked by chilly feelings or an actual rigor with intense pain in the abdomen. In typhoid fever, when the sensorium is benumbed, the onset may not be noticed. The pain is general and is usually intense and aggravated by movement and pressure. A position is taken which relieves the tension of the abdominal muscles, so that the patient lies on the back with the thighs drawn up and the shoulders elevated. The greatest pain is usually below the umbilicus, but in peritonitis from perforation of the stomach pain may be referred to the back, the chest, or the shoulder. The respiration is superficial—costal in type—as it is painful to use the diaphragm. For the same reason the action of coughing is restrained, and even the movements necessary for talking are limited. In this early stage the sensitiveness may be great and the abdominal muscles are often rigidly contracted. If the patient is at perfect rest the pain may be very slight, and there are instances in which it is not at all marked, and may, indeed, be absent.

The abdomen gradually becomes distended and tense and is tympanitic on percussion. The pulse is rapid, small, and hard, and often has a peculiar wiry quality. It ranges from 110 to 150. The temperature may rise rapidly after the chill and reach 104° or 105°, but the subsequent elevation is moderate. The tongue at first is white and moist, but subsequently becomes dry and often red and fissured. Vomiting is an early and prominent feature and causes great pain. The contents of the stomach are first ejected, then yellowish and bile-stained fluid, and finally a greenish and, in rare instances, a brownish-black liquid with slight fæcal odor. The bowels may be loose at the onset and then constipation follows. Frequent micturition may be present, less often retention. The urine is usually scanty and high-colored, and contains a large quantity of indican.

The appearance of the patient when these symptoms have fully developed is very characteristic. The face is pinched, the eyes are sunken, and the expression is very anxious. The constant vomiting of fluids causes a wasted appearance, and the hands sometimes present the washer-woman's skin. Except in cholera, we see the Hippocratic facies more frequently in this than in any other disease—"*a sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended, and parched; the color of the whole face being brown, black, livid, or lead-colored.*" There are one or two additional points about the abdomen. The tympany is usually excessive, owing to the great relaxation of the walls of the intestines by inflammation and exudation. The splenic dulness may be obliterated, the diaphragm pushed up, and the apex beat of the heart dislocated to the fourth interspace. The liver dulness may be greatly reduced, or may, in the mammary line, be obliterated. It has been claimed that this is a distinctive feature of perforative peritonitis, but on several occasions I have been able to demonstrate that the liver dulness in the middle and mammary line was obliterated by tympanites alone. In the axillary line, on the other hand, the liver dulness, though diminished, may persist. Pneumoperitonæum following perforation more certainly obliterates the hepatic dulness. In such cases the fluid effused produces a dulness in the lateral region; but with gas in the peritonæum, if the patient is turned on the left side, a clear note is heard beneath the seventh and eighth ribs in the axillary line.

Effusion of fluid—ascites—is usually present except in some acute, rapidly fatal cases. The flanks are dull on percussion. The dulness may be movable, though this depends altogether upon the degree of adhesions. There may be considerable effusion without either movable dulness or fluctuation. A friction-rub may be present, as first pointed out by Bright, but it is not nearly so common in acute as in certain forms of chronic peritonitis.

Course.—The acute diffuse peritonitis usually terminates in death. The most intense forms may kill within thirty-six or forty-eight hours; more commonly death results in four or five days, or the attack may be prolonged to eight or ten days. The pulse becomes more rapid, all the symptoms are aggravated, the vomiting persists and the patient usually dies in collapse with a falling temperature. Occasionally death occurs with great suddenness, owing, possibly, to paralysis of the heart.

Diagnosis.—In typical cases the severe pain at onset, the distention of the abdomen, the tenderness, the fever, the gradual development of effusion, collapse symptoms, and the vomiting give a characteristic picture. Careful inquiries should at once be made concerning the previous condition, from which a clew can often be had as to the starting-point of the trouble. In young adults a considerable proportion of all cases depends upon perforating appendicitis, and there may be an account of previous

attacks of pain in the iliac region, or of constipation alternating with diarrhœa. In women the most frequent causes are suppurative processes in the pelvic viscera, either associated with salpingitis, abscesses in the broad ligaments, or acute puerperal infection. Perforation of gastric ulcer is more common also in women. It is not always easy to determine the cause. Many cases come under observation for the first time with the abdomen distended and tender, and it is impossible to make a satisfactory examination. In such instances the pelvic organs should be examined with the greatest care. In typhoid fever, if the patient is conscious, the sudden onset of pain, the development of great meteorism, and the aggravation of the general symptoms indicate clearly what has happened. When the patient is in deep coma, on the other hand, the perforation may be overlooked. The following conditions are most apt to be mistaken for acute peritonitis:

(*a*) *Acute Entero-colitis.*—Here the pain and distention and the sensitiveness on pressure may be marked. The pain is more colicky in character, the diarrhœa is more frequent, and the collapse is more extreme.

(*b*) *The So-called Hysterical Peritonitis.*—This has deceived the very elect, as almost every feature of genuine peritonitis, even the collapse, may be simulated. The onset may be sudden, with severe pain in the abdomen, tenderness, vomiting, diarrhœa, difficulty in micturition, and the characteristic decubitus. Even the temperature may be elevated. There may be recurrence of the attack. A case has been reported by Bristowe in which four attacks occurred within a year, and it was not until special hysterical symptoms developed that the true nature of the trouble was suspected.

(*c*) *Obstruction of the bowel*, as already mentioned, may simulate peritonitis, both having pain, vomiting, tympanites, and constipation in common. It may for a couple of days really be impossible to make a diagnosis in the absence of a satisfactory history.

(*d*) *Rupture of an abdominal aneurism or embolism of the superior mesenteric artery* may cause symptoms which simulate peritonitis. In the latter, sudden onset with severe pain, the collapse symptoms, frequent vomiting, and great distention of the abdomen may be present.

(*e*) I have already referred to the fact that acute hæmorrhagic pancreatitis may be mistaken for peritonitis. Lastly, a ruptured tubal pregnancy may resemble acute peritonitis. A patient was admitted to my wards in an enfeebled condition, with a thready pulse, distended and tender abdomen, and signs of fluid. The attack had come on suddenly four days before, when she had been in perfect health. She looked pale, the blood count was taken and found below three millions per cubic centimetre, with leucocytosis, a condition rather indicating anæmia from hæmorrhage. The abdomen was tapped with a fine aspirator needle and a bloody fluid withdrawn. The diagnosis of probable ruptured tubal pregnancy was made and the patient was transferred to the gynæcological department, where laparotomy was performed and the ruptured tube removed.

II. PERITONITIS IN INFANTS.

Peritonitis may occur in the fœtus as a consequence of syphilis, and may lead to constriction of the bowel by fibrous adhesions.

In the new-born a septic peritonitis may extend from an inflamed cord. Distention of the abdomen, slight swelling and redness about the cord, and not infrequently jaundice are present. It is an uncommon event, and existed in only four of fifty-one infants dying of inflammation of the cord and septicæmia (Runge).

During childhood peritonitis develops from causes similar to those affecting the adult. Perforative appendicitis is common. Peritonitis following blows or kicks on the abdomen occurs more frequently at this period. In boys injury while playing foot-ball may be followed by diffuse peritonitis. A rare cause in children is extension through the diaphragm from an empyema. There are on record instances of peritonitis occurring in several children at the same school, and it has been attributed to sewer-gas poisoning. It was in investigating an epidemic of this kind at the Wandworth school, in London, that Anstie received the post-mortem wound of which he died.

III. LOCALIZED PERITONITIS.

The inflammation may be confined to the lesser peritonæum, particularly in cases of perforation of the stomach. A large air-containing abscess may form beneath the diaphragm, inducing the condition known as pyopneumothorax subphrenicus. More frequent is the circumscribed peritonitis due to inflammation of the appendix. If the vermiform process is free, adhesions take place which circumscribe the process. The most common situation is a localized abscess upon the psoas muscle, bounded by the cæcum on the right and the terminal portion of the ileum and its mesentery in front and on the left. The limitation may be complete, and post-mortem observation shows that healing follows in a large number of such cases. In other instances the localized peritonitis is more extensive and a large abscess cavity is gradually formed in the right iliac fossa, which may still be intraperitoneal, though shut off from the general sac. A more frequent cause of local peritonitis is inflammation about the uterus and Fallopian tubes, and here the primary disease is usually puerperal or gonorrhœal, less frequently tuberculous. The fimbriæ become adherent and closely matted to the ovary, and there is gradually produced a condition of thickening and matting of the parts in which the individual organs are scarcely recognizable. An acute process extending from this may involve only the pelvic membranes, being shut off from the general peritonæum by adhesions of the coils of the intestines.

IV. CHRONIC PERITONITIS.

The following varieties may be recognized: (*a*) **Local adhesive peritonitis**, a very common condition, which occurs particularly about the spleen, forming adhesions between the capsule and the diaphragm, about the liver, less frequently about the intestines and mesentery. Points of thickening or puckering on the peritonæum occur sometimes with union of the coils or fibrous bands. In a majority of such cases the condition is met accidentally post mortem. Two sets of symptoms may, however, be caused by these adhesions. When a fibrous band is attached in such a way as to form a loop or snare, a coil of intestine may pass through it. Thus, of the 295 cases of intestinal obstruction analyzed by Fitz, 63 were due to this cause. The second group is less serious and comprises cases with persistent abdominal pain of a colicky character, sometimes rendering life miserable. Instances of this kind have been successfully operated upon by Homans and H. A. Kelly.

(*b*) **Diffuse Adhesive Peritonitis.**—This is a consequence of an acute inflammation, either simple or tuberculous. The peritonæum is obliterated. On cutting through the abdominal wall, the coils of intestines are uniformly matted together and can neither be separated from each other nor can the visceral and parietal layers be distinguished. There may be thickening of the layers, and the liver and spleen are usually involved in the adhesions.

(*c*) **Proliferative Peritonitis.**—Apart from cancer and tubercle, which produce typical lesions of chronic peritonitis, the most characteristic form is that which may be described under this heading. The essential anatomical feature is great thickening of the peritoneal layers, usually without much adhesion. The cases are sometimes found with cirrhosis of the stomach. In one instance I found it in connection with a cirrhotic condition of the cæcum and the first part of the colon. In the inspection of a case of this kind there is usually moderate effusion, more rarely extensive ascites. The peritonæum is opaque-white in color, and everywhere thickened, often in patches. The omentum is usually rolled and forms a thickened mass transversely placed between the stomach and the colon. The peritonæum over the stomach, intestines, and mesentery is sometimes greatly thickened. The liver and spleen may simply be adherent, or there is a condition of chronic perihepatitis or perisplenitis, so that a layer of firm, almost gristly connective tissue of from one fourth to half an inch in thickness encircles these organs. Usually the volume of the liver is in consequence greatly reduced. The gastro-hepatic omentum may be constricted by this new growth and the calibre of the portal vein much narrowed. A serous effusion may be present. On account of the adhesions which form, the peritonæum may be divided into three or four different sacs, as is more fully described under the tuberculous peritonitis. In these cases the intestines are usually free, though the mesentery is greatly

shortened. There are instances of chronic peritonitis in which the mesentery is so shortened by this proliferative change that the intestines form a ball not larger than a cocoa-nut situated in the middle line, and after removal of the exudation can be felt as a solid tumor. The intestinal wall is greatly thickened and the mucous membrane of the ileum is thrown into folds like the valvulæ conniventes. This proliferative peritonitis is found frequently in the subjects of chronic alcoholism.

In all forms of chronic peritonitis a friction may be felt usually in the upper zone of the abdomen.

In some instances of chronic peritonitis the membrane presents numerous nodular thickenings, which may be mistaken for tubercles. They may be scattered in numbers on the membranes, and it may be extremely difficult, without the most careful microscopical examination, to determine their nature. J. F. Payne has described a case of this sort associated with disseminating growths throughout the liver which were not cancerous. It has been suggested that some of the cases of tuberculous peritonitis cured by operation have been of this nature, but histological examination would, as a rule, readily determine between the conditions. Miura, in Japan, has reported a case in which these nodules contained the ova of a parasite.

(*d*) **Chronic Hæmorrhagic Peritonitis.**—Blood-stained effusions in the peritonæum occur particularly in cancerous and tuberculous disease. There is a form of chronic inflammation analogous to the hæmorrhagic pachymeningitis of the brain. It was described first by Virchow, and is localized most commonly in the pelvis. Layers of new connective tissue form on the surface of the peritonæum with large wide vessels from which hæmorrhage occurs. This is repeated from time to time with the formation of regular layers of hæmorrhagic effusion. It is rarely diffuse, more commonly circumscribed.

V. NEW GROWTHS IN THE PERITONÆUM.

(*a*) **Tuberculous Peritonitis.**—This has already been considered.

(*b*) **Cancer of the Peritonæum.**—Although as a rule secondary to disease of the stomach, liver, or pelvic organs, cases of primary cancer are occasionally found. Secondary malignant peritonitis occurs in connection with all forms of cancer. It is usually characterized by a number of round tumors scattered over the entire peritonæum, sometimes small and miliary, at others large and nodular, with puckered centres. The disease most commonly starts from the stomach or the ovaries. The omentum is indurated, and, as in tuberculous peritonitis, forms a mass which lies transversely across the upper portion of the abdomen. Primary malignant disease of the peritonæum is extremely rare. Colloid has occurred, forming enormous masses, which in one case weighed over one hundred

pounds. Cancer of this membrane spreads, either by the detachment of small particles which are carried in the lymph currents and by the movements to distant parts, or by contact of opposing surfaces. It occurs more frequently in women than in men, and more commonly at the later period of life.

The *diagnosis* of cancer of the peritonæum is easy with a history of a local malignant disease; as when it occurs with ovarian tumor or with cancer of the pylorus. In cases in which there is no evidence of a primary lesion the diagnosis may be doubtful. The clinical picture is usually that of chronic ascites with progressive emaciation. There may be no fever. If there is much effusion nothing definite can be felt on examination. After tapping, irregular nodules or the curled omentum may be felt lying transversely across the upper portion of the abdomen. Unfortunately, this tumor upon which so much stress is laid occurs as frequently in tuberculous peritonitis and may be present in a typical manner in chronic proliferative form, so that in itself it has no special diagnostic value. Multiple nodules, if large, indicate cancer, particularly in persons above middle life. Nodular tuberculous peritonitis is most frequent in children. The presence about the navel of secondary nodules and indurated masses is more common in cancer. Inflammation, suppuration, and the discharge of pus from the navel rarely occur except in tuberculous disease. Considerable enlargement of the inguinal glands may be present in cancer. The nature of the fluid in cancer and in tubercle may be much alike. It may be hæmorrhagic in both; more often in the latter. The histological examination in cancer may show large multinuclear cells or groups of cells—the sprouting cell-groups of Foulis—which are extremely suggestive. The colloid cancer may produce a totally different picture; instead of ascitic fluid, the abdomen is occupied by the semi-solid gelatinous substance, and is firm, not fluctuating.

And, lastly, there are instances of echinococci in the peritonæum which may simulate cancer very closely. I have reported a case of this kind, in which the enlarged liver and the innumerable nodular masses in the peritonæum naturally led to this diagnosis.

VI. ASCITES (*Hydro-peritonæum*).

Definition.—The accumulation of serous fluid in the peritoneal cavity.

Etiology.—(1) **Local Causes.**—(*a*) Chronic inflammation of the peritonæum, either simple, cancerous, or tuberculous. (*b*) Portal obstruction in the terminal branches within the liver, as in cirrhosis, or by compression of the vein in the gastro-hepatic omentum, either by proliferative peritonitis, by new growths, or by aneurism. (*c*) Tumors of the abdomen. The solid growths of the ovaries may cause considerable ascites, which may

completely mask the true condition. The enlarged spleen in leukæmia, less commonly in malaria, may be associated with recurring ascites.

(2) **General Causes.**—The ascites is part of a general dropsy, the result of mechanical effects, as in heart-disease, chronic emphysema, and cirrhosis of the lung. In cardiac lesions the effusion is sometimes confined to the peritonæum, in which case it is due to secondary changes in the liver, or it has been suggested to be connected with a failure of the suction action of this organ, by which the peritonæum is kept dry. Ascites occurs also in the dropsy of Bright's disease, and in hydræmic states of the blood.

Symptoms.—A gradual uniform enlargement of the abdomen is the characteristic symptom of ascites. The physical signs are usually distinctive. (*a*) *Inspection.*—According to the amount of fluid the abdomen is protuberant and flattened at the sides. With large effusions, the skin is tense and may present the lineæ albicantes. Frequently the navel itself and the parts about it are very prominent. In many cases the superficial veins are enlarged and a plexus joining the mammary vessels can be seen. Sometimes it can be determined by pressure on these veins that the current is from below upward. In some instances, as in thrombosis or obliteration of the portal vein, these superficial abdominal vessels may be extensively varicose. About the navel in cases of cirrhosis there is occasionally a large bunch of distended veins, the so-called caput Medusæ.

(*b*) *Palpation.*—Fluctuation is obtained by placing the fingers of one hand upon one side of the abdomen and by giving a sharp tap on the opposite side with the other hand, when a wave is felt to strike as a definite shock against the applied fingers. Even comparatively small quantities of fluid may give this fluctuation shock. When the abdominal walls are thick or very fat, an assistant may place the edge of the hand or a piece of card-board in the front of the abdomen. A different procedure is adopted in palpating for the solid organs in case of ascites. Instead of placing the hand flat upon the abdomen, as in the ordinary method, the pads of the fingers only are placed lightly upon the skin, and then by a sudden depression of the fingers the fluid is displaced and the solid organ or tumor may be felt. By this method of "dipping" or displacement, as it is called, the liver may be felt below the costal margin, or the spleen, or sometimes solid tumors of the omentum or intestine.

(*c*) *Percussion.*—In the dorsal position with a moderate quantity of fluid in the peritonæum the flanks are dull, while the umbilical and epigastric regions, into which the intestines float, are tympanitic. This area of clear resonance may have an oval outline. Having obtained the lateral limit of the dulness on one side, if the patient then turns on the opposite side, the fluid gravitates to the dependent part and the uppermost flank is now tympanitic. In moderate effusions this movable dulness changes greatly in the different postures. Small amounts of fluid, probably under a litre, would scarcely give movable dulness, as the pelvis and the renal

regions hold a considerable quantity. In such cases it is best to place the patient in the knee-elbow position, when a dull note will be determined at the most dependent portion. By careful attention to these details mistakes are usually avoided.

The following are among the conditions which may be mistaken for dropsy: *Ovarian tumor*, in which the sac develops, as a rule, unilaterally, though when large it is centrally placed. The dulness is anterior and the resonance is in the flanks, into which the intestines are pushed by the cyst. Examination *per vaginam* may give important indications. In those rare instances in which gas develops in the cyst the diagnosis may be very difficult. Succussion has been obtained in such cases. A *distended bladder* may reach above the umbilicus. In such instances some urine dribbles away, and suspicion of ascites or a cyst is occasionally entertained. I once saw a trochar thrust into a distended bladder, which was supposed to be an ovarian cyst, and it is stated that John Hunter tapped a bladder, supposing it to be ascites. Such a mistake should be avoided by careful catheterization prior to any operative procedures. And lastly, there are large pancreatic or hydatid cysts in the abdomen which may simulate ascites.

Nature of the Ascitic Fluid.—Usually this is a clear serum, light yellow in the ascites of anæmia and Bright's disease, often darker in color in cirrhosis of the liver. The specific gravity is low, seldom more than 1·010 or 1·015. In the fluid of ovarian cysts the specific gravity is high, 1·020 or over. It is albuminous and sometimes coagulates spontaneously. Hæmorrhagic effusion usually occurs in cancer and tuberculosis, and occasionally in cirrhosis. I have already referred to the instances of hæmorrhagic effusion in connection with ruptured tubal pregnancy. A chylous, milky exudate is occasionally found. Busey has collected thirty-three cases from the literature. There are, as Quincke has pointed out, two distinct varieties, a fatty and a chylous, which may be distinguished by the microscope, as in the former there are distinct fat-globules. These cases have been sometimes connected with peritoneal or mesenteric cancer. In the true chylous ascites the fluid is turbid and milky. In some of the cases, as in Whitla's, a perforation of the thoracic duct has been found. The condition does not necessarily follow obliteration of the thoracic duct. Mild grades of chylous ascites, which are occasionally found clinically, may be due to the fact that the patient upon a milk diet has a permanent lipæmia, such as is present in young animals and in diabetics, in whom the liquor sanguinis is always fatty. Under such circumstances an exudate may contain enough of the molecular base of the chyle to produce turbidity of the fluid. Some of the cases have been associated with filariosis.

Treatment of the Previous Conditions.—(*a*) **Acute Peritonitis.**—Rest is enjoined upon the patient by the severe pain which follows the slightest movement, and he should be propped in the position

which gives him greatest relief. For the pain morphia should be injected hypodermically in full doses. In an adult it is better to give a third or half a grain at once, and subsequently at intervals repeat it in smaller doses, as are necessary. The action of the drug should be carefully watched and the patient should not be allowed to pass into such a degree of unconsciousness that he cannot be aroused. The respiration and the condition of the pupils also give valuable information. The amount of opium which has been given in certain instances is remarkable, and indicates a tolerance of the drug. The doses given by the late Alonzo Clark, of New York, may be truly termed heroic. Austin Flint notes that a patient under the care of this physician took "in the first twenty-four hours, of opium and the sulphate of morphia, a quantity equivalent to 106 grains of opium; in the second twenty-four hours she took 472 grains; on the third day, 236 grains; on the fourth day, 120 grains; on the fifth day, 54 grains; on the sixth day, 22 grains; on the seventh day, 18 grains; after which the treatment was suspended." It is unnecessary to use these enormous doses, as, even when the pain is most intense, from a third to a half grain of morphia every few hours will usually keep the patient thoroughly under the influence of the drug. In a robust, strong patient, seen at the outset, twenty leeches applied over the abdomen will give great relief.

Local applications—either hot turpentine stupes or cloths wrung out of ice-water—may be laid upon the abdomen. The patients sometimes declare that they are greatly relieved by the latter.

The question of the use of purgatives in peritonitis has of late been warmly discussed. Lawson Tait and other gynæcologists have used the saline purges with the greatest benefit in post-operation peritonitis. Theoretically it appears correct to give salines in concentrated form, which cause a rapid and profuse exosmosis of serum from the intestinal vessels, relieving the congestion and reducing the œdema, which is one important factor in causing the meteorism. It is also urged that the increased peristalsis prevents the formation of adhesions. In reading the reports of these successful cases, one is not always convinced, however, that peritonitis actually existed. Still, in cases of acute peritonitis due to extension or following operation or in septic conditions the judgment of many careful men is decidedly in favor of the use of salines. I cannot speak from personal experience on this question. The majority of cases of peritonitis which come under the care of the physician follow lesions of the abdominal viscera or are due to perforation of ulcer of the stomach, the ileum, or the appendix. In such cases, particularly in the large group of appendix cases, to give saline purgatives is, to say the least, most injudicious treatment. The safety of the patient lies in the restriction of the peristalsis and the localization of the inflammation, for which purpose opium alone is of service. In these instances rectal injections should be employed to relieve the large bowel. No symptom in acute peritonitis is more serious than

the tympanites, and none is more difficult to meet. The use of the long tube and injections containing turpentine may be tried. Drugs by the mouth cannot be retained.

For the vomiting, ice and small quantities of soda water may be employed. The patient should be fed on milk, but if the vomiting is distressing it is best not to attempt to give food by the mouth, but to use small nutrient enemata. In all cases of peritonitis it is best to have a surgeon in consultation early in the disease, as the question of operation may come up at any moment. I have already mentioned the conditions under which laparotomy is indicated in perforative appendicitis. The acute purulent cases, particularly those in which the streptococci occur, usually die; but although the results of operative interference in this form have not as yet been very brilliant, the condition, we must remember, is almost hopeless, and too often there has been unnecessary delay in calling in surgical aid. In the acute forms of tuberculous peritonitis operation appears to be more hopeful, but they are not always successful.

(*b*) **Chronic Peritonitis.**—For the cases of chronic proliferative peritonitis very little can be done. The treatment is practically that of ascites. In all these forms, when the distention becomes extreme, tapping is indicated. The treatment of tuberculous peritonitis has fallen largely into the hands of the surgeons, and the results in many cases are very good. According to the statistics of Maurange,* of 71 cases, 28 survived the operation for more than a year. Of 26 additional cases which I have collected,† 14 were dead at the time of the report. Within two years and three months there were six operations performed at the Johns Hopkins Hospital in tuberculous peritonitis, with four recoveries.

(*c*) **Ascites.**—The treatment depends somewhat on the nature of the case. In cirrhosis early and repeated tapping may give time for the establishment of the collateral circulation, and temporary cures have followed this procedure. Permanent drainage with Southey's tube, incision, and washing out the peritonæum have also been practised. In the ascites of heart and renal disease the cathartics are most satisfactory, particularly the bitartrate of potash, given alone or with jalap, and the large doses of salts given an hour before breakfast with as little water as possible. These sometimes cause rapid disappearance of the effusion, but they are not so successful in ascites as in pleurisy with effusion. The stronger cathartics may sometimes be necessary. The ascites forming part of the general anasarca of Bright's disease will receive consideration under another section.

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* Paris Thesis, 1889.

† On Tuberculous Peritonitis, Johns Hopkins Hospital Reports, 1890.

SECTION IV.

DISEASES OF THE RESPIRATORY SYSTEM.

I. DISEASES OF THE NOSE.

1. ACUTE CORYZA.

Acute catarrhal inflammation of the upper air-passages, popularly known as a "catarrh" or a "cold," is usually an independent affection, but may precede the development of another disease.

Etiology.—It prevails most extensively in the changeable weather of the spring and early winter, and may occur in epidemic form, many cases developing in a community within a few weeks. These outbreaks are very like, though less intense than the epidemic influenza, cases of which may begin with symptoms of ordinary coryza. The disease probably depends upon a micro-organism. Irritating fumes, such as those of iodine or ammonia, also may cause an acute catarrh of the nose.

Symptoms.—The patient feels indisposed, perhaps chilly, has slight headache, and sneezes frequently. In severe cases there are pains in the back and limbs. There is usually slight fever, the temperature rising to 101°. The pulse is quick, the skin is dry, and there are all the features of a feverish attack. At first the mucous membrane of the nose is swollen, "stuffed up," and the patient has to breathe through the mouth. A thin, clear, irritating secretion flows, and makes the edges of the nostrils sore. The mucous membrane of the tear-ducts is swollen, so that the eyes weep and the conjunctivæ are injected. With the nasal catarrh there is slight soreness of the throat and stiffness of the neck; the pharynx looks red and swollen, and sometimes the act of swallowing is painful. The larynx also may be involved, and the voice becomes husky or is even lost. If the inflammation extends to the Eustachian tubes there may be impairment of the hearing. Owing to the swelling of the nasal mucosa, the sense of smell and, in part, the sense of taste are lost. In more severe cases there are bronchial irritation and cough. Occasionally there is an outbreak of labial or nasal herpes. Usually within thirty-six hours the nasal secretion becomes turbid and more profuse, the swelling of the mucosa subsides, the patient gradually becomes able to breathe through the nostrils, and within

four or five days the symptoms disappear, with the exception of the increased discharge from the nose and upper pharynx. There are rarely any bad effects from a simple coryza. When the attacks are frequently repeated, the disease may become chronic.

The *diagnosis* is always easy, but caution must be exercised lest the initial catarrh of measles or severe influenza should be mistaken for the simple coryza.

Treatment.—Many cases are so mild that the patients are able to be about and to attend to their work. If there are fever and constitutional disturbance, the patient should be kept in bed and should take a simple fever mixture, and at night a drink of hot lemonade and a full dose of Dover's powder. Many persons find great benefit from the Turkish bath. For local treatment, particularly in the early stage, when the mucous membrane is swollen and there is a distressing sense of tightness and pain over the frontal sinuses, cocaine is very useful and sometimes gives immediate relief. The four per cent solution may be injected into the nostrils, or cotton-wool soaked in the solution may be inserted into them. Later, the snuff recommended by Ferrier is advantageous, composed, as it is, of morphia (gr. ij), bismuth (3 iv), acacia powder (3 ij). This may occasionally be blown or snuffed into the nostrils. Coryza is rarely serious in itself, but renders the subject more susceptible to other affections. The attacks should therefore never be slighted, and in young children and in the old especial care should be taken during convalescence.

II. CHRONIC NASAL CATARRH

(Rhinitis simplex; Rhinitis hypertrophica; Rhinitis atrophica).

In *simple chronic catarrh* there is increased irritability of the mucous membrane, particularly of the erectile tissue on the septum and turbinated bones. There is a tendency to frequent stoppage of one or both nostrils and the patient very easily catches cold. The secretion is at first clear and afterward thick and tenacious. The sense of smell is not specially disturbed at this stage. With the mirror the mucous membrane looks congested and swollen and the veins may be distended.

In *hypertrophic rhinitis*, which is usually a sequel of the former condition, the nasal passages are obstructed, chiefly by enlargement of the lower turbinated bodies and swelling of the mucous membrane of the septum. Very often there is hypertrophy of the adenoid tissue in the vault of the pharynx and of the mucous membrane about the orifices of the Eustachian tubes. The two conditions frequently go together as expressed in the designation, chronic naso-pharyngeal catarrh. The symptoms of this hypertrophic rhinitis may be local or general.

The most important local symptom is the obstruction of the passage of air through the nostrils, so that the patients become mouth-breathers.

During the day this may not be very distressing, but at night the mouth and throat get extremely dry and the sleep is disturbed. The voice becomes nasal in quality and in advanced cases, when the Eustachian tubes are obstructed, there may be deafness. It should ever be borne in mind by the practitioner that a very large proportion of all cases of deafness originate in chronic naso-pharyngeal catarrh. The general symptoms in these cases, particularly in children, are of the greatest importance, and have been considered more fully under chronic pharyngeal catarrh and mouth-breathing. Suffice it here to say that there is produced in children a characteristic facies, associated often with mental dulness and changes in the form of the thorax.

Atrophic rhinitis, which is also known under the names coryza fetida and ozæna, may be a sequence of the hypertrophic form. Ozæna is only a symptom, and is met with in many ulcerative conditions of the nostrils, particularly as a result of syphilis, foreign bodies, caries and necrosis of the bones, and glanders. Fortunately, the atrophic form by no means necessarily follows the hypertrophic stage. The cases are much more frequent in women than in men, and usually occur early in life. The mucous membrane is thin and covered with grayish crusts which, when removed, show a slightly excoriated surface, but true ulcers are rarely seen. The erectile tissue is completely atrophied by a process of slow connective-tissue growth, or, as J. N. Mackenzie calls it, a cirrhosis. The mucous membrane of the pharynx is usually dry and glazed.

The symptoms are most distinctive, owing to the horrible odor which comes from the nose, and of which, fortunately, the patient is himself unconscious, because the sense of smell is lost. The secretion, which is puriform, dries and forms large crusts, which are dislodged by picking or which gradually fall off. The cause of the offensive odor has been much discussed—whether it is due to a special organism or to specially favorable conditions for the growth and development of the germs of putrefaction. Probably the latter view is correct.

The *treatment* of hypertrophic rhinitis consists in the thorough cleansing of the nasal passages, the removal of the pharyngeal growths, and the reduction of the hypertrophied nasal mucosa. Operative procedures are necessary in a majority of the cases, and the practitioner should early call to his assistance the specialist. It is sad to think of the misery which has been entailed upon thousands of people owing to neglect of naso-pharyngeal catarrh by parents and physicians.

The treatment of atrophic rhinitis comes more properly under the special monographs.

III. AUTUMNAL CATARRH (*Hay Fever*).

An affection of the upper air-passages, often associated with asthmatic attacks, due to the action of certain stimuli upon a hypersensitive mucous membrane.

This affection was first described in 1819 by Bostock, who called it *catarrhus æstivus*. Morrill Wyman, of Cambridge, Mass., wrote a monograph on the subject, and described two forms, the "June cold," or "rose cold," which comes on in the spring, and the autumnal form which, in this country, does not develop until August and September, and never persists after a severe frost. Blakley studied its connection with the pollen of various grasses and flowers. The late George M. Beard made many careful observations on the disease. Until recently this form of catarrh was believed to result exclusively from the action of certain irritants on the mucous membrane of the nose, particularly the pollen of plants, which, as the experiments of Blakley showed, play an important *rôle* in the disease. Other emanations also may induce an attack, as in the case of the late Austin Flint, who was liable to coryza, or even asthma, if he slept on a certain sort of feather pillow. This, however, is only one factor in the disease. A second, most important one, was discovered in the condition of the nasal mucous membrane in these cases. Voltolini, of Breslau, in 1871, observed the cure of a case of asthma by the removal of a nasal polypus. Since that date the observations of Hack, in Germany, and particularly of Daly, of Pittsburg; Roe, of Rochester; John N. Mackenzie, of Baltimore; and Harrison Allen, of Philadelphia, have demonstrated the association of asthmatic attacks with nasal disease. Daly discovered that in a large proportion of the cases of hay asthma there was local disease of the mucous membrane of the nose, the cure of which rendered the patient insusceptible to conditions previously exciting the attacks. This has been abundantly confirmed. Still identical lesions exist in many people who never suffer with the disease, so that there must be a third factor, a neurotic constitution. In the etiology of hay fever, then, these three elements prevail—a nervous constitution, an irritable nasal mucosa, and the stimulus.

The disease affects certain families, particularly, it is said, those with a neurotic taint. The peculiarity may occur through several generations. It is certainly more common in the United States than in Europe, and much more common in the United States than in Canada. The United States Hay Fever Association now numbers thousands of members.

Dwellers in cities are more subject than residents in the country. The structural peculiarities of the nasal mucous membrane are those of hypertrophic rhinitis. Harrison Allen states that the inferior turbinated bones lie well above the floor of the nostrils, which renders the mucous membrane more liable to irritation from inhaled substances. Deflection of the septum, hypertrophy of the soft parts, and excessive hyperæsthesia, so that

the mere touch with a probe may be sufficient to induce an attack, are common conditions.

Symptoms.—These are, in a majority of the cases, very like those of ordinary coryza. There may, however, be much more headache and distress, and some patients become very low-spirited. Cough is a common symptom and may be very distressing. Paroxysms of asthma may develop, so like as to be indistinguishable from the ordinary bronchial form. The two conditions may indeed alternate, the patient having at one time an attack of common hay fever and at another, under similar circumstances, an attack of bronchial asthma. Of the immediate exciting causes of the attack, unquestionably in a majority of the cases coming on in the autumn there is an association with the presence of pollen in the atmosphere, but this is only one of a host of exciting causes. In certain persons the paroxysms may develop at any season from sudden changes in the temperature. An attack may even come on through association of ideas. The well-known experiment of J. N. Mackenzie, of inducing an attack in a susceptible person by offering her an artificial rose to smell, strikingly illustrates the neurotic element in the disease.

Treatment.—This may be comprised under three heads: First, since the disease appears in many instances to be a form of chronic neurosis, remedies which improve the stability of the nervous system may be employed—such as arsenic, phosphorus, and strychnia. Second, climatic. Dwellers in the cities of the Atlantic sea-board and of the Central States enjoy complete immunity in the Adirondacks and White Mountains. As a rule the disease is aggravated by residence in agricultural districts. The dry mountain air is unquestionably the best; there are cases, however, which do well at the seaside. Third, the thorough local treatment of the nose, particularly the destruction of the vessels and sinuses over the sensitive areas.

IV. EPISTAXIS.

Etiology.—Bleeding from the nose may result from local or constitutional conditions. Among local causes may be mentioned traumatism, picking or scratching the nose, new growths, and the presence of foreign bodies. In chronic nasal catarrh bleeding is not infrequent. The blood may come from one or both nostrils. The flow may be profuse after an injury, but is soon checked and is very rarely fatal. Occasionally profuse and fatal hæmorrhage occurs as a result of injury to the skull. In a remarkable case of this kind, coming on some weeks after the receipt of the injury, I found that there had been a fracture across the sphenoid bone and an erosion had taken place into the carotid artery, just where it runs closest to the sphenoidal sinuses. The young man had completely recovered from the effects of the injury, and the fatal hæmorrhage took place as he was stooping over to wash his face.

Among general conditions with which nose-bleeding is associated, the following are the most important: It occurs with great frequency in growing children, particularly about the age of puberty; more frequently in the delicate than in the strong and vigorous.

Epistaxis is a very common event in persons of so-called plethoric habits. It is stated sometimes to precede, or to indicate a liability to, apoplexy, but this is very doubtful.

In venous engorgement, due to heart or pulmonary disease, epistaxis is not common and there may be a most extreme grade of cyanosis without its occurrence. In balloon and mountain ascensions, in the very rarefied atmosphere, hæmorrhage from the nose is a common event. In hæmophilia the nose ranks first of the mucous membranes from which bleeding arises. It occurs in all forms of chronic anæmias. It precedes the onset of certain fevers, more particularly typhoid, with which it seems associated in a special manner. Vicarious epistaxis has been described in cases of suppression of the menses. Lastly, it is said to be brought on by certain psychical impressions, but the observations on this point are not trustworthy. The blood in epistaxis results from capillary oozing or diapedesis. The mucous membrane is deeply congested and there may be small ecchymoses. The bleeding area is usually in the respiratory portion of one nostril and upon the cartilaginous septum.

Symptoms.—Slight hæmorrhage is not associated with any special features. When the bleeding is protracted the patients have the more serious manifestations of loss of blood. In the slow dripping which takes place in some instances of hæmophilia, there may be formed a remarkable blood tumor projecting from one nostril and extending even below the mouth.

Death from ordinary epistaxis is very rare. The more blood is lost, the greater is the tendency to clotting with spontaneous cessation of the bleeding.

The *diagnosis* is usually easy. One point only need be mentioned; namely, that bleeding from the posterior nares occasionally occurs during sleep and the blood trickles into the pharynx and may be swallowed. If vomited, it may be confounded with hæmatemesis; or, if coughed up, with hæmoptysis.

Treatment.—In a majority of the cases the bleeding ceases of itself. Various simple measures may be employed, such as holding the arms above the head, the application of ice to the nose, or the injection of cold or hot water into the nostrils. Astringents, such as zinc, alum, or tannin, may be used; and the old-fashioned and sometimes successful remedy, a cobweb, may be introduced into the nostrils. If the bleeding comes from an ulcerated surface, an attempt should be made to apply chromic acid or to cauterize. If the bleeding is at all severe and obstinate, the posterior nares should be plugged. Ergot may be given internally or hypodermically.

II. DISEASES OF THE LARYNX.

I. ACUTE CATARRHAL LARYNGITIS.

This may come on as an independent affection or in association with general catarrh of the upper respiratory passages.

Etiology.—Many cases are due to catching cold or to overuse of the voice; others develop in consequence of the inhalation of irritating gases. It may occur in the general catarrh associated with influenza and measles. Very severe laryngitis is excited by traumatism, either injuries from without or the lodgment of foreign bodies. It may be caused by the action of very hot liquids or corrosive poisons.

Symptoms.—There is a sense of tickling referred to the larynx; the cold air irritates and, owing to the increased sensibility of the mucous membrane, the act of inspiration may be painful. There is a dry cough, and the voice is altered. At first it is simply husky, but soon phonation becomes painful, and finally the voice may be completely lost. In adults the respirations are not increased in frequency, but in children dyspnœa is not uncommon and may occur in spasmodic attacks. If much œdema accompanies the inflammatory swelling, there may be urgent dyspnœa.

The laryngoscope shows a swollen and tumefied mucous membrane of the larynx, particularly the ary-epiglottidean folds. The vocal cords have lost their smooth and shining appearance and are reddened and swollen. Their mobility also is greatly impaired, owing to the infiltration of the adjoining mucous membrane and of the muscles. A slight mucoid exudation covers the parts. The constitutional symptoms are not severe. There is rarely much fever, and in many cases the patient is not seriously ill. Occasionally cases come on with greater intensity, the cough is very distressing, deglutition is painful, and there may be urgent dyspnœa.

Diagnosis.—There is rarely any difficulty in determining the nature of a case if a satisfactory laryngoscopic examination can be made. The severer forms may simulate œdema of the glottis. When the loss of voice is marked, the case may be mistaken for one of nervous aphonia, but the laryngoscope would decide the question at once. Much more difficult is the diagnosis of acute laryngitis in children, particularly in the very young, in whom it is so hard to make a proper examination. From ordinary laryngismus it is to be distinguished by the presence of fever, the mode of onset, and particularly the coryza and the previous symptoms of hoarseness or loss of voice. Membranous laryngitis may at first be quite impossible to differentiate, but in a majority of cases of this affection there are patches on the pharynx and early swelling of the cervical glands. The symptoms, too, are much more severe.

Treatment.—Rest of the larynx should be enjoined, so far as phonation is concerned. In cases of any severity the patient should be kept

in bed. The room should be at an even temperature and the air saturated with moisture. Early in the disease, if there is much fever, aconite and citrate of potash can be given, and for the irritating painful cough a full dose of Dover's powder at night. An ice-bag externally often gives great relief.

II. CHRONIC LARYNGITIS.

Etiology.—The cases usually follow repeated acute attacks. The most common causes are overuse of the voice, particularly in persons whose occupation necessitates shouting in the open air. The constant inhalation of irritating substances, as tobacco-smoke, may also cause it.

Symptoms.—The voice is usually hoarse and rough and in severe cases may be almost lost. There is usually very little pain; only the unpleasant sense of tickling in the larynx, which causes a frequent desire to cough. With the largyngoscope the mucous membrane looks swollen, but much less red than in the acute condition. In association with the granular pharyngitis, the mucous glands of the epiglottis and of the ventricles may be involved.

Treatment.—The nostrils should be carefully examined, since in some instances chronic laryngitis is associated with and even dependent upon obstruction to the free passage of air through the nose. Local application must be made directly to the larynx, either with a brush or by means of a spray. Among the remedies most recommended are the solutions of nitrate of silver, chlorate of potash, perchloride of zinc, and tannic acid. Insufflations of bismuth are sometimes useful.

Among directions to be given are the avoidance of heated rooms and loud speaking, and abstinence from tobacco and alcohol. The throat should not be too much muffled, and morning and evening the neck should be sponged with cold water.

III. ŒDEMATOUS LARYNGITIS.

Etiology.—Œdema of the glottis, or, more correctly, of the structures which form the glottis, is a very serious affection which is met with (*a*) as a rare sequence of ordinary acute laryngitis, whether due to cold or to the application of irritants. (*b*) In chronic diseases of the larynx, as syphilis or tubercle. (*c*) In severe inflammatory diseases like diphtheria, in erysipelas of the neck, and in various forms of cellulitis. (*d*) Occasionally in the acute infectious diseases—scarlet fever, typhus, or typhoid. In Bright's disease, either acute or chronic, there may be a rapidly developing œdema. The connection with Bright's disease has been disputed and is certainly rare. I have met with two instances, one in scarlatinal

nephritis and the other in chronic interstitial nephritis. Both cases proved fatal in a short time.

Symptoms.—There is dyspnœa, increasing in intensity, so that within an hour or two the condition becomes very serious. There is sometimes marked stridor in respiration. The voice becomes husky and disappears. The laryngoscope shows enormous swelling of the epiglottis, which can sometimes be felt with the finger or even seen when the tongue is strongly depressed with a spatula. The ary-epiglottidean folds are the seat of the chief swelling and may almost meet in the middle line. Occasionally the œdema is below the true cords.

The diagnosis is rarely difficult, inasmuch as even without the laryngoscope the swollen epiglottis can be seen or felt with the finger. The disease is very fatal.

Treatment.—An ice-bag should be placed on the larynx and the patient given ice to suck. If the symptoms are urgent, the throat should be sprayed with a strong solution of cocaine, and the swollen epiglottis scarified. If relief does not follow, tracheotomy should immediately be performed. The high rate of mortality is due to the fact that this operation is as a rule too long delayed.

IV. MEMBRANOUS LARYNGITIS (*Croup*).

Inflammation of the larynx, with membranous exudation occurs: (1) As a simple, non-specific, non-contagious affection, local in its nature, and not occurring in epidemics. It is unquestionably a rare disease. (2) As an effect of diphtheria, in which the disease may be limited to the larynx, but most commonly is associated with exudation on the pharynx or tonsils.

Etiology.—Membranous croup is now regarded by many authorities as always diphtheritic, and while it must be acknowledged that this is so in the great majority of instances, there are cases, few in number, it is true, in which it is not possible to assign this origin. The question may be settled by the presence or absence of Loeffler's bacillus, which is a definite criterion of diphtheritic pseudo-membrane. At the Montreal General Hospital, which received annually an exceptionally large number of cases of diphtheria, we were in the habit of regarding all the laryngeal cases as true diphtheria, even when no patches could be seen on the tonsils. On several occasions, in cases of this kind, I have been able to demonstrate post mortem that the exudation had extended at the back of the tonsils or on the posterior pillars of the fauces. On the other hand, twice at the Infant's Home I saw cases, sporadic in their nature, coming on suddenly without much fever, with extensive fibrinous exudation, necessitating tracheotomy, but without a trace of pharyngeal exudation. Although the conditions were most favorable for the spread of

the infection in the Home, no other cases occurred. Provisionally, at any rate, I still hold that there is a separate independent affection, a non-contagious membranous croup. Yet I am willing to acknowledge that the large majority of the cases of fibrinous laryngitis are due to the poison of diphtheria. It is particularly desirable that a bacteriological examination should be made of the membrane in the former class of cases.

The disease affects young children, particularly between the ages of two and six. Cases under two and over seven are very rare. Statistics show that boys are more often attacked than girls Cases occur occasionally with scarlet fever and measles.

Morbid Anatomy.—On inspection of the larynx of a child dead of membraneous croup, the *rima* is seen filled with mucus or with a shreddy material which, when washed off carefully, leaves the mucosa covered by a thin grayish-yellow membrane, which may be uniform or in patches. It covers the ary-epiglottic folds and the true cords, and may be continued into the ventricles or even into the trachea. Above, it may involve the epiglottis. It varies much in consistency. I have seen fatal cases in which the exudation was not actually membranous, but rather friable and granular. It may form a thick, even stratified membrane, which fills the entire glottis. The exudation may extend down the trachea and into the bronchi, and may pass beyond the epiglottis to the fauces. Usually it can be readily stripped off from the mucous membrane of the larynx and leaves exposed the swollen and injected mucosa. On examination it is seen that the fibrinous material has involved chiefly the epithelial lining and has not greatly infiltrated the subjacent tissues.

Symptoms.—Naturally, the clinical symptoms are almost identical in the non-specific and specific forms of membranous laryngitis.

The affection begins like an acute laryngitis with slight hoarseness and rough cough, to which the term croupy has been applied. After these symptoms have lasted for a day or two with varying intensity, the child suddenly becomes worse, usually at night, and there are signs of impeded respiration. At first the difficulty in breathing is paroxysmal, due probably to more or less spasm of the muscles of the glottis. Soon the dyspnœa becomes continuous, inspiration and expiration become difficult, particularly the latter, and with the inspiratory movements the epigastrium and lower intercostal spaces are retracted. The voice is husky and may be reduced to a whisper. The color gradually changes and the imperfect aëration of the blood is shown in the lividity of the lips and finger-tips. Restlessness comes on and the child tosses from side to side, vainly trying to get breath. Occasionally, in a severer paroxysm, portions of membrane are coughed out. The fever in non-specific membranous laryngitis is rarely high and the condition of the child is usually very good at the time of the onset. The pulse is always increased in frequency and if cyanosis be present is small. In favorable cases the dyspnœa is not very urgent, the color of the face remains good, and after one or two paroxysms the child goes to sleep and

wakes in the morning, perhaps without fever and feeling comfortable. The attack may recur the following night with greater severity. In unfavorable cases the dyspnœa becomes more and more urgent, the cyanosis deepens, the child, after a period of intense restlessness, sinks into a semi-comatose state, and death finally occurs from poisoning of the nerve centres by carbon dioxide. In diphtheritic laryngitis the onset is usually less sudden and is preceded by a longer period of indisposition. As a rule, there are pharyngeal symptoms. The constitutional disturbance, too, is more severe, the fever higher, and there may be swelling of the glands of the neck. Inspection of the fauces may show the presence of false membranes on the pillars or on the tonsils. This, however, is held by some not to be an invariable evidence of the diphtheritic nature of the inflammation. Fagge held that non-contagious membranous croup may spread upward from the larynx just as diphtheritic inflammation is in the habit of spreading downward from the fauces. Ware, of Boston, whose essay on croup is perhaps the most solid contribution to the subject made in this country, reported the presence of exudate in the fauces in 74 out of 75 cases of croup. These observations were made prior to 1840, during periods in which diphtheria was not epidemic to any extent in Boston. In protracted cases pulmonary symptoms may develop, which are sometimes due to the difficulty in expelling the muco-pus from the tubes; in others, the false membrane extends into the trachea and even into the bronchial tubes. During the paroxysm the vesicular murmur is scarcely audible, but the laryngeal stridor may be loudly communicated along the bronchial tubes.

Diagnosis.—Membranous laryngitis must be distinguished from ordinary simple laryngitis and from certain spasmodic affections. Simple catarrhal laryngitis rarely induces such severe symptoms, occurs more suddenly, nearly always at night, and the hoarseness and implication of the voice are not nearly so marked. The presence of preceding symptoms is one of the most important diagnostic distinctions between the false and the true croup. By hoarseness, dyspnœa, and signs of membrane on the fauces or tonsils the existence of membranous laryngitis may be definitely determined. Occasionally simple laryngitis induces swelling sufficient to cause marked dyspnœa and hoarseness and may, indeed, prove fatal. Of course, true membranous laryngitis may follow the catarrhal form. In laryngismus the attack comes on suddenly and is not associated with either cough or hoarseness. The child is seized with a difficulty in breathing; the inspirations are crowing in character, and the dyspnœa rapidly becomes urgent, so that symptoms of suffocation supervene, sometimes within less than a minute; the spasm then relaxes and the child appears to be in its normal condition. It is most commonly met with in rickety children.

The diagnosis between diphtheritic and non-diphtheritic membranous laryngitis is by no means easy, and, as mentioned above, many excellent authorities hold the diseases to be identical. The following are the chief points of distinction, which refer to general rather than to local conditions: The

non-specific affection generally begins in the larynx and the fauces are but slightly, if at all, affected. It is not infectious. Cases develop in institutions under circumstances most favorable to the spread of the disease, but other children are not attacked. It has none of the serious asthenic symptoms of diphtheria, and it is not followed by paralysis. It occurs almost exclusively in very young children, whereas diphtheritic laryngitis is not at all uncommon in adults.

Prognosis.—True croup, whether simple or diphtheritic, with a mortality of from sixty to eighty per cent, is an extremely fatal disease. When it attacks healthy children and is not secondary to some febrile affection, the outlook is more hopeful. Even a very limited exudation may prove fatal. On several occasions, in performing post-mortems in fatal cases, I have been astonished to find such a slight involvement of the larynx; in some instances scarcely more than a granular exudation covering the cords and folds. A fatal result is almost inevitable when the disease extends to the bronchi.

Treatment.—As the cases rarely come under observation until the membrane is formed, the main medicinal indication is to favor its separation. The air of the room should be saturated with moisture from an atomizer and the throat should be sprayed with lime-water.

In young children topical application to the larynx itself is extremely difficult and in many instances impossible. Good results have followed the passage of a sponge-probang with a strong solution of nitrate of silver. It is an easy matter to recommend such measures, but very difficult to carry them out. The administration of a brisk emetic will sometimes bring away portions of the false membrane; ipecacuanha or the turpeth mineral is the most suitable. Of late years there has been a return to the mercurial treatment of membranous laryngitis, but I have not seen such results from its use as would justify a recommendation of it. Continuous hot applications to the throat are usually much more grateful than the ice-bag, so highly recommended by some practitioners. With the first indication of defective aëration of the blood it is well to let the child inhale oxygen, which may be conveniently passed into a tent made of sheets on the bed.

In very many cases the obstruction reaches such a grade that the propriety of intubation or tracheotomy is raised. One great advantage of the former is that it may be suggested at an earlier stage with more likelihood of gaining the consent of the parents.

The statistics of tracheotomy are not very satisfactory, as only a fourth to a third of the cases recover.

The general treatment of these cases is of great importance. In the first place the child should be isolated, since it is often impossible to say whether the case is specific or not. Much of the success in the case depends upon careful nursing. There is no disease which requires greater care, coolness, and judgment on the part of the attendants. The diet

should consist of milk and beef-juices. Water should be given freely to the child, and if the pulse shows signs of failing, stimulants should at once be administered. The extreme restlessness calls for anodynes, but they must be administered with great care; bromide and chloral are to be preferred to opium. In cases in which the dyspnœa comes on in paroxysms, as if due to spasm, I have seen great benefit follow the inhalation of chloroform.

V. SPASMODIC LARYNGITIS (*Laryngismus stridulus*).

Spasm of the glottis is met with in many affections of the larynx, but there is a special disease in children which has received the above-mentioned names.

Etiology.—A purely nervous affection, without any inflammatory condition of the larynx, it occurs in children between the ages of six months and three years, and is most commonly seen in connection with rickets. It is also associated with tetany. Often the attack comes on when the child has been crossed or scolded. Mothers sometimes call the attacks "passion fits" or attacks of "holding the breath." It was supposed at one time that they were associated with enlargement of the thymus, and they therefore received the name of *thymic asthma*.

The actual condition of the larynx during a paroxysm is a spasm of the adductors, but the precise nature of the influences causing it is not yet known, whether centric or reflex from peripheral irritation. The disease is not so common in America as in England.

Symptoms.—The attacks may come on either in the night or in the day; often just as the child awakes. There is no cough, no hoarseness, but the respiration is arrested and the child struggles for breath, the face gets congested, and then, with a sudden relaxation of the spasm, the air is drawn into the lungs with a high-pitched crowing sound, which has given to the affection the name of "child-crowing." Convulsions may occur during an attack or there may be carpo-pedal spasms. Death may, but rarely does, occur during the attack. With the cyanosis the spasm relaxes and respiration begins. The attacks may recur with great frequency throughout the day.

Treatment.—The gums should be carefully examined and, if swollen and hot, freely lanced. The bowels should be carefully regulated and as these children are usually delicate or rickety nourishing diet and cod-liver oil should be given. By far the most satisfactory method of treatment is the cold sponging. In severe cases, two or three times a day the child should be placed in a warm bath and the back and chest thoroughly sponged for a minute or two with cold water. Since learning this practice from Ringer, at the University Hospital, I have seen many cases in which it proved successful. It may be employed when the child is in

a paroxysm, though if the attack is severe and the lividity is great it is much better to dash cold water into the face. Sometimes the introduction of the finger far back into the throat will relieve the spasm.

Spasmodic croup, believed to be a functional spasm of the muscles of the larynx, is an affection seen most commonly between the the ages of two and five years. According to Trousseau's description, the child goes to bed well, and about midnight or in the early morning hours awakes with oppressed breathing, harsh, croupy cough, and perhaps some huskiness of voice. The oppression and distress for a time are very serious, the face is congested, and there are signs of approaching cyanosis. The attack passes off abruptly, the child falls asleep and awakes the next morning feeling perfectly well. These attacks may be repeated for several nights in succession, and usually cause great alarm to the parents. Whether this is entirely a functional spasm is, I think, doubtful. There are instances in which the child is somewhat hoarse through the day, and has slight catarrhal symptoms and a brazen, croupy cough. There is probably slight catarrhal laryngitis with it. These cases are not infrequently mistaken for true croup, and parents are sometimes unnecessarily disturbed by the serious view which the physician takes of the case. Too often the poor child, deluged with drugs, is longer in recovering from the treatment than he would be from the disease. To allay the spasm a whiff of chloroform may be administered, which will in a few moments give relief, or the child may be placed in a hot bath. A prompt emetic, such as zinc or wine of ipecac, will usually relieve the spasm, and is specially indicated if the child has overloaded the stomach through the day.

VI. TUBERCULOUS LARYNGITIS.

Etiology.—Tubercles may develop primarily in the laryngeal mucosa, but in the great majority of cases the affection is secondary to pulmonary tuberculosis, in which it is met with in a variable proportion of from eighteen to thirty per cent. Males are more frequently affected than females, possibly, as Bosworth suggested, because they are more frequently subject to catarrhal laryngitis, which is undoubtedly a predisposing cause. Laryngitis may occur very early in pulmonary tuberculosis. There may be well-marked involvement of the larynx with signs of very limited trouble at one apex. These are cases which, in my experience, run a very unfavorable course.

Morbid Anatomy.—The mucosa is at first swollen and presents scattered tubercles, which seem to begin in the neighborhood of the blood-vessels. By their fusion small tuberculous masses arise, which caseate and finally ulcerate, leaving shallow irregular losses of substance. The ulcers are usually covered with a grayish exudation, and there is a general thickening of the mucosa about them, which is particularly marked upon the

arytenoids. The ulcers may erode the true cords and finally destroy them, and passing deeply may cause perichondritis with necrosis and occasionally exfoliation of the cartilages. The disease may extend laterally and involve the pharynx, and downward over the mucous membrane covering the cricoid cartilage toward the œsophagus. Above, it may reach the posterior wall of the pharynx, and in rare cases extend to the fauces and tonsils. The epiglottis may be entirely destroyed. There are rare instances in which cicatricial changes go on to such a degree that stenosis of the larynx is induced, a remarkable specimen of which I saw some years ago with J. Solis-Cohen.

Symptoms.—The first indication is slight huskiness of the voice, which finally deepens to hoarseness, and in advanced stages there may be complete loss of voice. There is something very suggestive in the early hoarseness of tuberculous laryngitis. My attention has frequently been directed to the lungs simply by the quality of the voice.

The cough is in part due to involvement of the larynx. Early in the disease it is not very troublesome, but when the ulceration is extensive it becomes husky and ineffectual. Of the symptoms of laryngeal tuberculosis, none is more aggravating than the dysphagia, which is met with particularly when the epiglottis is involved, and when the ulceration has extended to the pharynx. There is no more distressing or painful complication in phthisis. In instances in which the epiglottis is in great part destroyed, with each attempt to take food there are distressing paroxysms of cough, and even of suffocation.

With the laryngoscope there is seen early in the disease a pallor of the mucous membrane, which also looks thickened and infiltrated, particularly that covering the arytenoid cartilages. The tuberculous ulcers are very characteristic. They are broad and shallow, with gray bases and ill-defined outlines. The vocal cords are infiltrated and thickened, and ulceration is very common.

The diagnosis of tuberculous laryngitis is rarely difficult, as it is usually associated with well-marked pulmonary disease. In case of doubt some of the secretion from the base of an ulcer should be removed and examined for bacilli.

Treatment.—Physicians pay scarcely sufficient attention to the laryngeal complications of consumption. The ulcers should be sprayed and kept thoroughly cleansed. Solutions of tannic acid, nitrate of silver, or sulphide of zinc may be employed. The insufflation, two or three times a day, of a powder of iodoform, with morphia, after thoroughly cleansing the ulcers with a spray, relieves the pain in a majority of the cases. Cocaine (four per cent solution) applied with the atomizer will often enable the patient to swallow his food comfortably. There are, however, distressing cases of extensive laryngeal and pharyngeal ulceration in which even cocaine loses its good effects. When the epiglottis is lost the difficulty in swallowing becomes very great. Wolfenden states that this may be obvi-

ated if the patient hangs his head over the side of the bed and sucks milk through a rubber tubing from a mug placed on the floor.

VII. SYPHILITIC LARYNGITIS.

Syphilis attacks the larynx with great frequency. It may result from the inherited disease or be a secondary or tertiary manifestation of the acquired form.

Symptoms.—In secondary syphilis there is occasionally erythema of the larynx, which may go on to definite catarrh, but has nothing characteristic. The process may proceed to the formation of superficial whitish ulcers, usually symmetrically placed on the cords or ventricular bands. Mucous patches and condylomata are rarely seen. The symptoms are practically those of slight loss of voice with laryngeal irritation, as in the simple catarrhal form.

The tertiary laryngeal lesions are numerous and very serious. True gummata, varying in size from the head of a pin to a small nut, develop in the submucous tissue most commonly at the base of the epiglottis. They go through the changes characteristic of these structures and may either break down, producing extensive and deep ulceration, or—and this is more characteristic of syphilitic laryngitis—in their healing form a fibrous tissue which shrinks and produces stenosis. The ulceration is apt to extend deeply and involve the cartilage, inducing necrosis and exfoliation, and even hæmorrhage from erosion of the arteries. Œdema may suddenly prove fatal. The cicatrices which follow the sclerosis of the gummata or the healing of the ulcers produce great deformity. The epiglottis, for instance, may be tied down to the pharyngeal wall or to the epiglottic folds, or even to the tongue; and eventually a stenosis results, which may necessitate tracheotomy.

The laryngeal symptoms of inherited syphilis have the usual course of these lesions and appear either early, within the first five or six months, or after puberty; most commonly in the former period. Of 76 cases, J. N. Mackenzie found that 63 occurred within the first year. The gummatous infiltration leads to ulceration, most commonly of the epiglottis and in the ventricles, and the process may extend deeply and involve the cartilage. Cicatricial contraction may also occur.

The diagnosis of syphilis of the larynx is rarely difficult, since it occurs most commonly in connection with other symptoms of the disease. For special details the manuals of laryngology should be consulted.

Treatment.—The administration of constitutional remedies is the most important, and under mercury and iodide of potassium the local symptoms may rapidly be relieved. The tertiary laryngeal manifestations are always serious and difficult to treat. The deep ulceration is specially

hard to combat, and the cicatrization may necessitate tracheotomy, or the gradual dilatation, as practised by Schroetter.

III. DISEASES OF THE BRONCHI.

I. ACUTE BRONCHITIS.

Acute catarrhal inflammation of the bronchial mucous membrane is a very common disease, rarely serious in healthy adults, but very fatal in the old and in the young, owing to associated pulmonary complications. It is bilateral and affects either the larger and medium sized tubes or the smaller bronchi, in which case it is known as capillary bronchitis.

We shall speak only of the former, as the latter is part and parcel of broncho-pneumonia.

Etiology.—Acute bronchitis is a common sequence of catching cold, and is often nothing more than the extension downward of an ordinary coryza. It occurs most frequently in the changeable weather of early spring and late autumn. Its association with cold is well indicated by the popular expression "cold on the chest." It may prevail as an epidemic apart from influenza, of which it is an important feature.

Acute bronchitis is associated with many other affections, notably measles. It is by no means rare at the onset of typhoid fever and malaria. It is present also in asthma and whooping-cough. The bronchitis of Bright's disease, gout, and heart-disease is usually a chronic form. It attacks persons of all ages, but most frequently the young and the old. There are individuals who have a special disposition to bronchial catarrh, and the slightest exposure is apt to bring on an attack. Persons who live an out-of-door life are usually less subject to the disease than those who follow sedentary occupations.

The affection is probably microbic, though we have as yet no definite evidence upon this point.

Morbid Anatomy.—The mucous membrane of the trachea and bronchi is reddened, congested, and covered with mucus and muco-pus, which may be seen oozing from the smaller bronchi, some of which are dilated. The finer changes in the mucosa consist in desquamation of the ciliated epithelium, swelling and œdema of the submucosa, and infiltration of the tissue with leucocytes. The mucous glands are much swollen.

Symptoms.—The symptoms of an ordinary "cold" accompany the onset of an acute bronchitis. The coryza extends to the tubes, and may also affect the larynx, producing hoarseness, which in many cases is marked. A chill is rare, but there is invariably a sense of oppression, with heaviness and languor and pains in the bones and back. In mild cases there is scarcely any fever, but in severer forms the range is from 101° to 103°.

The bronchial symptoms set in with a feeling of tightness and rawness beneath the sternum and a sensation of oppression in the chest. The cough is rough at first, cutting and sore, and often of a ringing character. It comes on in paroxysms which rack and distress the patient extremely. During the severe spells the pain may be very intense beneath the sternum and along the attachments of the diaphragm. At first the cough is dry, but in a few days the secretion becomes muco-purulent and abundant, and finally purulent. With the loosening of the cough great relief is experienced. The sputum is made up largely of pus-cells, with a variable number of the large round alveolar cells, many of which contain carbon grains, while others have undergone the myelin degeneration.

Physical Signs.—The respiratory movements are not greatly increased in frequency unless the fever is high. There are instances, however, in which the breathing is rapid and when the smaller tubes are involved there is dyspnœa. On palpation the bronchial fremitus may often be felt. On auscultation in the early stage, piping sibilant râles are everywhere to be heard. They are very changeable, and appear and disappear with coughing. With the relaxation of the bronchial membranes and the greater abundance of the secretion, the râles change and become mucous and bubbling in quality.

The *course* of the disease depends on the conditions under which it develops. In healthy adults, by the end of a week the fever subsides and the cough loosens. In another week or ten days convalescence is fully established. In young children the chief risk is in the extension of the process downward. In measles and whooping-cough, the ordinary bronchial catarrh is very apt to descend to the finer tubes, which become dilated and plugged with muco-pus, inducing areas of collapse, and finally broncho-pneumonia. This extension is indicated by changes in the physical signs. Usually at the base the râles are subcrepitant and numerous and there may be areas of defective resonance and of feeble or distant tubular breathing. In the aged and debilitated there are similar dangers if the process extends from the larger to the smaller tubes. In old age the bronchial mucosa is less capable of expelling the mucus, which is more apt to sag to the dependent parts and induce dilatation of the tubes with extension of the inflammation to the contiguous air-cells.

The *diagnosis* of acute bronchitis is rarely difficult. Although the mode of onset may be brusque and perhaps simulate pneumonia, yet the absence of dulness and blowing breathing, and the general character of the bronchial inflammation, renders the diagnosis simple. The complication of broncho-pneumonia is indicated by the greater severity of the symptoms, particularly the dyspnœa, the defective color, and the physical signs.

Treatment.—In mild cases, household measures suffice. The hot foot-bath, or the warm bath, a drink of hot lemonade, and a mustard plaster on the chest will often give relief. For the dry, racking cough, the symptom most complained of by the patient, Dover's powder is the best

remedy. It is a popular belief that quinine, in full doses, will check an oncoming cold in the chest, but this is doubtful. It is a common custom when persons feel the approach of a cold to take a Turkish bath, and though the tightness and oppression may be relieved by it, there is in a majority of the cases great risk. Some of the severest cases of bronchitis which I have seen have followed this initial Turkish bath. No doubt, if the person could go to bed directly from the bath, its action would be beneficial, but there is great risk of catching additional "cold" in going home from the bath. Relief is obtained from the unpleasant sense of rawness by keeping the air of the room saturated with moisture, and in this dry stage the old-fashioned mixture of the wines of antimony and ipecacuanha with liquor ammoniæ acetatis and nitrous ether is useful. If the pulse is very rapid, tincture of aconite may be given, particularly in the case of children. For the cough, when dry and irritating, opium should be freely used in the form of Dover's powder. Of course, in the very young and the aged care must be exercised in the use of opium, particularly if the secretions are free; but for the distressing, irritative cough, which keeps the patient awake, no remedy can take its place As the cough loosens and the expectoration is more abundant, the patient becomes more comfortable. In this stage it is customary to ply the patient with expectorants of various sorts. Though useful occasionally, they should not be given as a matter of routine. A mixture of squills, ammonia, and senega is a favorite one with many practitioners at this stage.

In the acute bronchitis of children, if the amount of secretion is large and difficult to expectorate, or if there is dyspnœa and the color begins to get dusky, an emetic (a tablespoonful of ipecac wine) should be given at once and repeated if necessary.

II. CHRONIC BRONCHITIS.

Etiology.—This affection may follow repeated attacks of acute bronchitis, but it is most commonly met with in chronic lung affections, heart-disease, gout, and renal disease. It is frequent in the aged; the young rarely are affected. Climate and season have an important influence. It is the winter cough of the old man, which recurs with regularity as the weather gets cold and changeable.

Morbid Anatomy.—The bronchial mucosa presents a great variety of changes, depending somewhat upon the disease with which chronic bronchitis is associated. In some cases the mucous membrane is very thin, so that the longitudinal bands of elastic tissue stand out prominently. The tubes are dilated and the muscular and glandular tissues are atrophied and the epithelium in great part shed.

In other instances the mucosa is thickened, granular, and infiltrated. There may be ulceration, particularly of the mucous follicles. Bronchial

dilatations are not uncommon and emphysema is a constant accompaniment.

Symptoms.—In the form met with in old men, associated with emphysema, gout, or heart-disease, the chief symptoms are as follows: Shortness of breath, which may not be noticeable except on exertion. The patients "puff and blow" on going up hill or up a flight of stairs. This is due not so much to the chronic bronchitis itself as to associated emphysema or even to cardiac weakness. They complain of no pain. The cough is variable, changing with the weather and with the season. During the summer they may remain free, but each succeeding winter the cough comes on with severity and persists. There may be only a spell in the morning, or the chief distress is at night. The sputum in chronic bronchitis is very variable. In cases of the so-called dry catarrh there is no expectoration. Usually, however, it is abundant, muco-purulent, or distinctly purulent in character. There are instances in which the patient coughs up for years a thin fluid sputum. There is rarely fever. The general health may be good and the disease may present no serious features apart from the liability to incidental emphysema and bronchiectasy. In many cases it is an incurable affection. Patients improve and the cough disappears in the summer time only to return during the winter months.

Physical Signs.—The chest is usually distended, the movements are limited, and the condition is often that which we see in emphysema. The percussion note is clear or hyperresonant. On auscultation, expiration is prolonged and wheezy and rhonchi of various sorts are heard—some high-pitched and piping, others deep-toned and snoring. Crepitation is common at the bases.

Clinical Varieties.—The description just given is of the ordinary chronic bronchitis which occurs in connection with emphysema and heart-disease and in many elderly men. There are certain forms which merit special description: (*a*) On several occasions I have met with a form of *chronic bronchitis*, particularly in women, which comes on between the ages of twenty and thirty and may continue indefinitely without serious impairment of the health. In one case, a lady of fifty, with a phthisical family history, began to cough when she was twenty-five, and since then has had more or less cough every day without intermission. It has not seriously impaired her health, though she has never been strong. Once or twice she has had attacks of eczema. The cough is chiefly in the morning, is apt to be brought on by too much conversation, and is quite independent of the weather. The daily amount of expectoration is not great, rarely more than from four to six ounces. It is muco-purulent in character. The examination of the chest is negative—no emphysema, no râles. I have met several such instances which seem to form a type of chronic bronchitis, though it is difficult to say upon what the condition depends.

(*b*) *Bronchorrhœa.*—Excessive bronchial secretion is met with under several conditions. It must not be mistaken for the profuse expectoration

of bronchiectasy. The secretion may be very liquid and watery—*bronchorrhœa serosa*. More commonly, it is purulent though thin, and with greenish or yellow-green masses. It may be thick and uniform. This profuse bronchial secretion is usually a manifestation of chronic bronchitis and may lead to dilatation of the tubes and ultimately to fetid bronchitis. In the young the condition may persist for years without impairment of health and without apparently damaging the lungs.

(*c*) *Putrid Bronchitis.*—Fetid expectoration is met with in connection with bronchiectasis, gangrene, abscess, or with decomposition of secretions within phthisical cavities and in an empyema which has perforated the lung. There are instances in which, apart from any of these states, the expectoration has a fetid character. The sputa are abundant, usually thin, grayish white in color, and they separate into an upper fluid layer capped with frothy mucus and a thick sediment in which may sometimes be found dirty yellow masses the size of peas or beans—the so-called Dittrich's plugs. The affection is very rare apart from the above-mentioned conditions. In severe cases it leads to changes in the bronchial walls, pneumonia, and often to abscess or gangrene. Metastatic brain abscess has followed putrid bronchitis in a certain number of cases.

(*d*) *Dry Catarrh.—Catarrhe sec* of Laennec is a not uncommon form, characterized by paroxysms of coughing of great intensity, with little or no expectoration. It is usually met with in elderly persons with emphysema, and is one of the most chronic and obstinate of all varieties of bronchitis.

Treatment.—By far the most satisfactory method of treating the recurring winter bronchitis is change of climate. Removal to a southern latitude may prevent the onset. Southern France, southern California, and Florida furnish winter climates in which the subjects of chronic bronchitis live with the greatest comfort. All cases of prolonged bronchial irritation are benefited by change of air.

The first endeavor in treating a case of chronic bronchitis is to ascertain if possible whether there are constitutional or local affections with which it is associated. In many instances the urine is found to be highly acid, perhaps slightly albuminous, and the arteries are stiff. In the form associated with this condition, sometimes called gouty bronchitis, the attacks seem related to the defective renal elimination, and to this condition the treatment should be first directed. In other instances there are heart-disease and emphysema. In the form occurring in old men much may be done in the way of prophylaxis. Septuagenarians should read Oliver Wendell Holmes's * "De Senectute" with reference to the care of the health and the avoidance of catching cold. He lays stress upon the importance of the daily study of the thermometer and barometer. There is no doubt that with prudence even in our changeable winter weather much may be

* Over the Tea-cups, Boston, 1890.

done to prevent the onset of chronic bronchitis. Woolen undergarments should be used and especial care should be taken in the spring months not to change them for lighter ones before the warm weather is established.

Cure is seldom effected by medicinal remedies. There are instances in which iodide of potassium acts with remarkable benefit, and it should always be given a trial in cases of paroxysmal bronchitis of obscure origin. When the secretion is excessive the muriate of ammonia is perhaps the most useful. Stimulating expectorants are contra-indicated. When the heart is feeble, the combination of digitalis and strychnia is very beneficial. Turpentine, the old-fashioned remedy so warmly recommended by the Dublin physicians, has in many quarters fallen undeservedly into disuse. Terebene in capsules is a useful substitute because it is more easily taken. Of other balsamic remedies, sandalwood, the compound tincture of benzoin, copaiba, balsam of Peru or tolu may be used. Inhalations are often very useful. If fetor be present, carbolic acid in the form of spray (twenty to thirty per cent solution) will lessen the odor, or thymol (1 to 1,000). In full-blooded men, when venous engorgement exists and shortness of breath, the abstraction of twenty to thirty ounces of blood will afford prompt relief.

III. BRONCHIECTASIS.

Etiology.—Dilatation of the bronchi occurs under the following conditions: (1) As a congenital defect or anomaly. Such cases are extremely rare, commonly unilateral. Grawitz has described the condition as *bronchiectasis universalis*. Welch has met an instance in a young girl. (2) In connection with inflammation of the bronchi, particularly when this leads to weakness of the walls with the accumulation of secretion. Under this category come the dilatation met with in chronic bronchitis and emphysema, the dilated bronchi in chronic phthisis, in the catarrhal pneumonias of children, and particularly the dilitation which results from the presence of foreign bodies in the air-tubes or from pressure, as of an aneurism on one bronchus. (3) In extreme contraction of the lung tissue, whether due to interstitial pneumonia or to compression by pleural adhesions, bronchial dilatation is a common though not a constant accompaniment.

Unquestionably the weakening of the bronchial wall is the most important, probably the essential, factor in inducing bronchiectasy, since the wall is then not able to resist the pressure of air in severe spells of coughing and in straining. In some instances the mere weight of the accumulated secretion may be sufficient to distend the terminal tubules, as is seen in compression of a bronchus by aneurism.

Morbid Anatomy.—Two chief forms are recognized—the *cylindrical* and the *saccular*—which may exist together in the same lung. The

condition may be general or partial. Universal bronchiectasis is always unilateral. It occurs in rare congenital cases and is occasionally seen as a sequence of interstitial pneumonia. The entire bronchial tree is represented by a series of sacculi opening one into the other. The walls are smooth and possibly without ulceration or erosion except in the dependent parts. The lining membrane of the sacculi is usually smooth and glistening. The dilatations may form large cysts immediately beneath the pleura. Intervening between the sacculi is a dense cirrhotic lung tissue. The partial dilatations—the saccular and cylindrical—are common in chronic phthisis, particularly at the apex, in chronic pleurisy at the base, and in emphysema. Here the dilatation is more commonly cylindrical, sometimes fusiform. The bronchial mucous membrane is much involved and sometimes there is a narrowing of the lumen. Occasionally one meets with a single saccular bronchiectasy in connection with chronic bronchitis or emphysema. Some of these look like simple cysts, with smooth walls, without fluid contents.

Histologically the bronchi which are the seat of dilatation show important changes. In the large, smooth dilatations the cylindrical is replaced by a pavement epithelium. The muscular layer is stretched, atrophied, and the fibres separated; the elastic tissue is also much stretched and separated. In the large saccular bronchiectasies and in some of the cylindrical forms, due to retained secretions, the lining membrane is ulcerated. The contents of some of the larger bronchiectatic cavities are horribly fetid.

Symptoms.—In the limited dilatations of phthisis, emphysema, and chronic bronchitis, the symptoms are in great part those of the original disease, and the condition often is not suspected during life.

In extensive saccular bronchiectasy the characters of the cough and expectoration are distinctive. The patient will pass the greater part of the day without any cough and then in a severe paroxysm will bring up a large quantity of sputum. Sometimes change of the position will bring on a violent attack, probably due to the fact that some of the secretion flows from the dilatation to a normal tube. The daily spell of coughing is usually in the morning. The expectoration is in many instances very characteristic. It is grayish or grayish brown in color, fluid, purulent, with a peculiar acid, sometimes fetid, odor. Placed in a conical glass, it separates into a thick granular layer below and a thin mucoid intervening layer above, which is capped by a brownish froth. Microscopically it consists of pus-corpuscles, often large crystals of fatty acids, which are sometimes in enormous numbers over the field and arranged in bunches. Hæmatoidin crystals are sometimes present. Elastic fibres are seldom found except when there is ulceration of the bronchial walls. Tubercle bacilli are not present. In some cases the expectoration is very fetid and has all the characters of those described under fetid bronchitis. Nummular expectoration, such as comes from phthisical cavities, is not

common. Hæmorrhage may occur, but in my experience it has been rare. Abscess of the brain has in a few instances followed the bronchiectasis. Rheumatoid affections may develop (Gerhardt).

The *diagnosis* is not possible in a large number of the cases. In the extensive sacculated forms, unilateral and associated with interstitial pneumonia or chronic pleurisy, the diagnosis is easy. There is contraction of the side, which in some instances is not at all extreme. The cavernous signs may be chiefly at the base and may vary according to the condition of the cavity, whether full or empty. There may be the most exquisite amphoric phenomena and loud resonant râles. The condition persists for years and is not inconsistent with tolerably active life. The patients frequently show signs of marked embarrassment of the pulmonary circulation. There is cyanosis on exertion, the finger-tips are clubbed, and the nails incurved. A condition very difficult to distinguish from bronchiectasy is a limited pleural cavity communicating with a bronchus.

Treatment.—Medical treatment is not satisfactory, since it is impossible to heal the cavity. I have practised the injection of antiseptic fluids in some instances with benefit. In suitable cases drainage of the cavities may be attempted, particularly if the patient is in fairly good condition. For the fetid secretion turpentine may be given or terebene, and inhalations used of carbolic acid or thymol. In extreme cases it is very difficult to get rid of the offensive odor.

IV. BRONCHIAL ASTHMA.

Asthma is a term which has been applied to various conditions associated with dyspnœa—hence the names cardiac and renal asthma—but its use should be limited to the affection known as bronchial or spasmodic asthma.

Etiology.—All writers agree that there is in a majority of cases of bronchial asthma a strong neurotic element. Many regard it as a neurosis in which, according to one view, spasm of the bronchial muscles, according to the other, turgescence of the mucosa, results from disturbed innervation, pneumogastric or vaso-motor. Of the numerous theories the following are the most important:

(1) That it is due to spasm of the bronchial muscles, a theory which has perhaps the largest number of adherents. The original experiments of C. J. B. Williams, upon which it is largely based, have not, however, been confirmed of late years.

(2) That the attack is due to swelling of the bronchial mucous membrane—fluctionary hyperæmia (Traube), vaso-motor turgescence (Weber), diffuse hyperæmic swelling (Clark).

(3) That in many cases it is a special form of inflammation of the smaller bronchioles—*bronchiolitis exudativa* (Curschmann). Other theo-

ries which may be mentioned are that the attack depends on spasm of the diaphragm or on reflex spasm of all the inspiratory muscles.

As already mentioned, the so-called hay fever is an affection which has many resemblances to bronchial asthma, with which the attacks may alternate. In the suddenness of onset and in many of their features these diseases have the same origin and differ only in site, as suggested by Sir Andrew Clark and now generally acknowledged by specialists. Making due allowance for anatomical differences, if the structural changes occurring in the nasal mucous membrane during an attack of hay fever were to occur also in various parts of the bronchial mucosa, their presence there would afford a complete and adequate explanation of the facts observed during a paroxysm of bronchial asthma (Clark). With this statement I fully agree, but the observations of Curschmann have directed attention to a feature in asthma which has been neglected; namely, that in a majority of the cases it is associated with an exudation, such as might be supposed to come from a turgescent mucosa and which is of a very characteristic and peculiar character. The hyperæmia and swelling of the mucosa and the extremely viscid, tenacious mucus explain well the hindrance to inspiration and expiration and also the quality of the râles.

Some general facts with reference to etiology may be mentioned. The affection sometimes runs in families, particularly those with irritable and unstable nervous systems. The attack may be associated with neuralgia or, as Salter mentions, even alternate with epilepsy. Men are more frequently affected than women. The disease often begins in childhood and sometimes lasts until old age. One of its most striking peculiarities is the *bizarre* and extraordinary variety of circumstances which at times induce a paroxysm. Among these local conditions climate or atmosphere are most important. A person may be free in the city and invariably suffer from an attack when he goes into the country, or into one special part of the country. Such cases are by no means uncommon. Breathing the air of a particular room or a dusty atmosphere may bring on an attack. Odors, particularly of flowers and of hay, or emanations from animals, as the horse, dog, or cat, may at once cause an outbreak. Fright or violent emotion of any sort may bring on a paroxysm. Uterine and ovarian troubles were formerly thought to induce attacks and may do so in rare instances. Diet, too, has an important influence, and in persons subject to the disease severe paroxysms may be induced by overloading the stomach, or by taking certain articles of food. Chronic cases, in which the attacks recur year after year, gradually become associated with emphysema, and every fresh "cold" induces a paroxysm. And lastly, many cases of bronchial asthma are associated with affections of the nose, particularly with hypertrophic rhinitis and nasal polypi. According to some specialists of large experience, all cases of bronchial asthma have some affection of the upper air-passages, but I am convinced from personal observation that this is erroneous. Still physicians must acknowledge the debt which we

owe to Voltolini, Hack, Daly, Roe, and others who have shown the close connection which exists between affections of the nose and many cases of bronchial asthma.

Briefly stated then, bronchial asthma is a neurotic affection, characterized by hyperæmia and turgescence of the mucosa of the smaller bronchial tubes and a peculiar exudate of mucin. The attacks may be due to direct irritation of the bronchial mucosa or may be induced reflexly, by irritation of the nasal mucosa, and indirectly, too, by reflex influences, from stomach, intestines, or genital organs.

Symptoms.—Premonitory sensations precede some attacks, such as chilly feeling, a sense of tightness in the chest, flatulence, passage of a large quantity of urine, or great depression of spirits. Nocturnal attacks are common. After a few hours' sleep, the patient is aroused with a distressing sense of want of breath and a feeling of great oppression in the chest. Soon the respiratory efforts become violent, all the accessory muscles are brought into play, and in a few minutes the patient is in a paroxysm of the most intense dyspnœa. The face is pale, the expression anxious, speech is impossible, and in spite of the most strenuous inspiratory efforts very little air enters the lungs. Expiration is prolonged and also wheezy. The number of respirations, however, is not much increased. The asthmatic fit may last from a few minutes to several hours. When severe, the signs of defective aëration soon appear, the face becomes bedewed with sweat, the pulse is small and quick, the extremities get cold, and just as the patient seems to be at his worst, the breathing begins to get easier, and often with a paroxysm of coughing relief is obtained and he sinks exhausted to sleep. The relief may be but temporary and a second attack may soon come on. In a majority of the cases even in the intervals between the asthmatic fits the respiration is somewhat embarrassed. The cough is at first very tight and dry and the expectoration is expelled with the greatest difficulty.

The physical signs during an attack are very characteristic. On inspection the thorax looks enlarged, barrel-shaped, and is fixed, the amount of expansion being altogether disproportionate to the intensity of the inspiratory movements. The diaphragm is lowered and moves but slightly. Inspiration is short and quick, expiration prolonged. Percussion may not reveal any special difference, but there is sometimes marked hyperresonance, particularly in cases which have had repeated attacks.

On auscultation, with inspiration and expiration, there are innumerable sibilant and sonorous râles of all varieties, piping and high-pitched, low-pitched and grave. Later in the attack there are moist râles.

The *sputum* in bronchial asthma is quite distinctive, unlike that which occurs in any other affection. Early in the attack it is brought up with great difficulty and is in the form of rounded gelatinous masses, the so-called "*perles*" of Laennec. Though ball-like, they can be unfolded and really represent moulds in mucus of the smaller tubes. The entire expec-

toration may be made up of these somewhat translucent-looking pellets, floating in a small quantity of thin mucus. Some of them are opaque. Often with the naked eye a twisted spiral character can be seen, particularly if the sputum is spread on a glass with a black background. Microscopically, many of these pellets have a spiral structure, which renders them among the most remarkable bodies met with in sputum. It is not a little curious that they should have been practically overlooked until described a few years ago by Curschmann. Under the microscope the spirals are of two forms. In one there is simply a twisted, spirally arranged mucin, in which are entangled cells, derived probably from the smaller bronchi and alveoli, often in all stages of fatty degeneration. The twist may be loose or tight. The second form is much more peculiar. In the centre of a tightly coiled skein of mucin fibrils with a few scattered cells is a filament of extraordinary clearness and translucency, probably composed of transformed mucin. As Curschmann suggests, these spirals are doubtless formed in the finer bronchioles and constitute the product of an acute bronchiolitis. It is difficult to explain their spiral nature. I do not know of any observations upon the course of the currents produced by the ciliated epithelium in the bronchi, but it is quite possible that their action may be rotatory, in which case, particularly when combined with spasm of the bronchial muscles, it is possible to conceive that the mucus formed in the tube might be compelled to assume a spiral form. Within two or three days the sputum changes entirely in character; it becomes muco-purulent and Curschmann's spirals are no longer to be found. They occur in all instances of true bronchial asthma in the early period of the attack. There are, in addition, in many cases, the pointed, octahedral crystals described by Leyden and sometimes called asthma crystals. They are identical with the crystals found in the semen and in the blood in leukæmia. At one time they were supposed, by their irritating character, to induce the paroxysms.

The *course* of the disease is very variable. In severe attacks the paroxysms recur for three or four nights or even more, and in the intervals and during the day there may be wheezing and cough. Early in the disease the patient may be free in the morning, without cough or much distress, and the attacks may appear at first to be of a purely nervous character. In the long-standing cases emphysema almost invariably develops, and while the pure asthmatic fits diminish in frequency the chronic bronchitis and shortness of breath become aggravated.

We have no knowledge of the morbid anatomy of true asthma. Death during the attack is unknown. In long-standing cases the lesions are those of chronic bronchitis and emphysema.

Treatment.—The asthmatic attack usually demands immediate and prompt treatment, and remedies should be administered which experience has shown are capable of relieving the condition of the bronchial mucosa. A few whiffs of chloroform will produce prompt though temporary relaxa-

tion. In a child with very severe attacks, resisting all the usual remedies, the treatment by chloroform gave immediate and finally permanent relief. Perles of nitrite of amyl may be broken on the handkerchief or from two to five drops of the solution may be placed upon cotton-wool and inhaled. Strong stimulants given hot or a dose of spirits of chloroform in hot whisky will sometimes induce relaxation. More permanent relief is given by the hypodermic injection of morphia or of morphia and cocaine combined. In obstinate and repeatedly recurring attacks this has proved a very satisfactory plan. The sedative antispasmodics, such as belladonna, henbane, stramonium, and lobelia, may be given in solution or used in the form of cigarettes. Nearly all the popular remedies either in this form or in pastilles contain some of the plant of the order *solanaceæ*, with nitrate or chlorate of potash. Excellent cigarettes are now manufactured and asthmatics try various sorts, since one form benefits one patient, another form another patient. Nitre paper made with a strong solution of nitrate of potash is very serviceable. Filling a room with the fumes of this paper prior to retiring will sometimes ward off a nocturnal attack. I have known several patients to whom tobacco smoke inhaled was quite as potent as the prepared cigarettes.

The use of compressed air in the pneumatic cabinet is very beneficial; oxygen inhalations may be also tried. In preventing the recurrence of the attacks there is no remedy so useful as iodide of potassium, which sometimes acts like a specific. From ten to twenty grains three times a day is usually sufficient.

Particular attention should be paid to the diet of asthmatic patients. A rule which experience generally compels them to make is to take the heavy meals in the early part of the day and not retire to bed before gastric digestion is completed. As the attacks are often induced by flatulency, the carbohydrates should not be allowed. Coffee is a more suitable drink than tea. In respect to climate it is very difficult to lay down rules for asthmatics. The patients are often much better in the city than in the country. The high and dry altitudes are certainly more beneficial than the sea-shore; but in protracted cases, with emphysema as a secondary complication, the rarefied air of high altitudes is not advantageous. In young persons I have known a residence for six months in Florida or southern California to be followed by prolonged freedom from attacks.

V. FIBRINOUS BRONCHITIS.

An acute or chronic affection, characterized by the formation in certain of the bronchial tubes of fibrinous casts, which are expelled in paroxysms of dyspnœa and cough.

In several diseases fibrinous moulds of the bronchi are formed, as in diphtheria and croup (with extension into the trachea and bronchi), in

pneumonia, and occasionally in phthisis—conditions which, however, have nothing to do with true fibrinous bronchitis. These casts are not to be confounded with the blood-casts which occur occasionally in hæmoptysis.

Etiology.—Nothing is known of its causation. It occurs more frequently in males. It is met with at all periods of life, but is more common between the ages of twenty and forty. It has been known to attack several members of the same family. Cases have been described occurring together as if due to some endemic influence (Pichini). The cases are rare, particularly in hospital practice. The attacks occur most commonly in the spring months. An association with tuberculosis has been frequently noted. Model, in an article from Bäumler's clinic, states that tuberculosis was present in ten of twenty-one post-mortems. It has been met with also in connection with skin-diseases, such as pemphigus, impetigo, and herpes. The attacks appeared to be related in some cases to the menstrual period. Several instances have been described with heart-disease, but it seems probable that in all these conditions the connection was not causal.

Symptoms.—Acute cases are rare. They may set in with high fevers, rigors, severe paroxysms of cough, and perhaps with hæmoptysis. The clinical picture resembles acute bronchitis, and only the expulsion of the membranous casts gives the characteristic features to the case. It is much more serious than the chronic form and fatal termination is not uncommon. N. S. Davis has reported two fatal cases. In some of the acute cases there has been affection of the tonsils, and it is possible that the disease may have been truly diphtheritic in character and due to extension of the membrane into the trachea and bronchi. The casts in these cases are not only more extensive, but they also do not present the laminated structure characteristic of true plastic bronchitis.

A patient may have a single attack without any recurrence, but in the chronic form the attacks come on at varying intervals and the disease may last for ten or even twenty years. Instances are on record in which the paroxysms have occurred at definite intervals for many months. The attacks may recur weekly or a period of a year or more may intervene. The onset is marked by bronchitic symptoms, not necessarily with fever. The cough becomes distressing and paroxysmal in character; the sputa may be blood-stained and the patient brings up rounded, ball-like masses, which, when disentangled, are found to be moulds of bronchi; the hæmorrhage may be profuse. In one of the two cases which I have seen it invariably accompanied the attack, and the whitish dendritic casts of the tubes were always entangled in the blood and clots. Urgent dyspnœa and cyanosis may be present in severe attacks. The *physical* signs are those of a severe bronchitis. It may occasionally be possible to determine the weakened or suppressed breath sounds in the affected territory and there may be deficient expansion or even retraction of the chest wall in a corresponding area, but this is in reality very difficult, and twice prior to the expulsion of the casts I failed to determine by physical examination the affected region.

As mentioned, the casts are usually rolled up and mixed with mucus or blood. When unravelled in water they present a complete mould of a secondary or tertiary bronchus with its ramifications. The size of the cast may vary with different attacks, but, as has often been noticed, the form and size may be identical at each attack as if precisely the same bronchial area was involved each time. The casts are hollow, laminated, the size of the lumen varying with the number and thickness of the laminæ. Sometimes they are almost solid. Transverse sections show a beautiful concentric arrangement. The fibrin appears in places to retain its fibrillary structure; in others, as in diphtheritic membrane, it has undergone the hyaline transformation. Leucocytes are imbedded in the meshes. In the centre, particularly in the smaller casts, it is not uncommon to see alveolar epithelium with numerous carbon particles. Leyden's crystals are sometimes found and occasionally Curschmann's spirals.

The pathology of the disease is obscure. The membrane is identical with that to which the term croupous is applied, and the obscurity relates not so much to the mechanism of the production, which is probably the same as in other mucous surfaces, as to the curious limitation of the affection to certain bronchial territories and the remarkable recurrence at stated or irregular intervals throughout a period of many years.

In the acute cases the *treatment* should be that of ordinary acute bronchitis. We know of nothing which can prevent the recurrence of the attacks in the chronic form. In the uncomplicated cases there is rarely any danger during the paroxysm, even though the symptoms may be most distressing and the dyspnœa and cough very severe. Inhalations of ether, steam, or atomized lime-water aid in the separation of the membranes. Pilocarpine might be useful, as in some instances it increases the bronchial secretion. The employment of emetics may be necessary, and in some cases they are effective in promoting the removal of the casts.

IV. DISEASES OF THE LUNGS.

I. CIRCULATORY DISTURBANCES IN THE LUNGS.

Congestion.—There are two forms of congestion of the lungs—active and passive.

(1) *Active Congestion of the Lungs.*—Much doubt and confusion still exist on this subject. French writers, following Woillez, regard it as an independent primary affection (*maladie de Woillez*), and in their dictionaries and text-books allot much space to it. English and American authors more correctly regard it as a symptomatic affection. Active fluxion to the lungs occurs with increased action of the heart, and when very hot air or irritating substances are inhaled. In diseases which interfere

locally with the circulation the capillaries in the adjacent unaffected portions may be greatly distended. The importance, however, of this collateral fluxion, as it is called, is probably exaggerated. In a whole series of pulmonary affections there is this associated congestion—in pneumonia, bronchitis, pleurisy, and tuberculosis.

The symptoms of active congestion of the lungs are by no means definite. The description given by Woillez and by other French writers is of an affection which is difficult to recognize from anomalous or larval forms of pneumonia. The chief symptoms described are initial chill, pain in the side, dyspnœa, moderate cough, and temperature from 101° to 103°. The physical signs are defective resonance, feeble breathing, sometimes bronchial in character, and fine râles. A majority of clinical physicians would undoubtedly class such cases under inflammation of the lung. In many epidemics the abnormal and larval forms are specially prevalent. This is no doubt the condition to which Porcher, of Charlestown, called attention a short time ago as a "hitherto undescribed affection of the lungs."

The occurrence of an intense and rapidly fatal congestion of the lung, following extreme heat or cold or sometimes violent exertion, is recognized by some authors. Renforth, the oarsman, is said to have died from this cause during the race at Halifax. Leuf has described cases in which, in association with drunkenness, exposure, and cold, death occurred suddenly, or within twenty-four hours, and the only lesion found has been an extreme, almost hæmorrhagic, congestion of the lungs. It is by no means certain that in these cases death really occurs from pulmonary congestion in the absence of specific statements with reference to the coronary arteries. Several times in sudden death from disease of these vessels I have seen great engorgement of the lungs though not the extreme grade mentioned by Leuf. I have no personal knowledge of cases such as he describes.

(2) *Passive Congestion.*—Two forms of this may be recognized, the mechanical and the hypostatic.

(*a*) Mechanical congestion occurs whenever there is an obstacle to the return of the blood to the heart. It is a common event in many affections of the left heart. The lungs are voluminous, russet brown in color, cutting and tearing with great resistance. On section they show at first a brownish-red tinge, and then the cut surface, exposed to the air, becomes rapidly of a vivid red color from oxidation of the abundant hæmoglobin. This is the condition known as *brown induration* of the lung. Histologically it is characterized by (α) great distention of the alveolar capillaries; (β) increase in the connective-tissue elements of the lung; (γ) the presence in the alveolar walls of many cells containing altered blood-pigment, (δ) in the alveoli numerous epithelial cells containing blood-pigment in all stages of alteration, which are also found in great numbers in the sputum.

It occasionally happens that this mechanical hyperæmia of the lung results from pressure of tumors. So long as compensation is maintained the mechanical congestion of the lung in heart-disease does not produce any symptoms, but with enfeebled heart action the engorgement becomes marked and there are dyspnœa, cough, and expectoration, with the characteristic alveolar cells.

(*b*) Hypostatic congestion. In fevers and adynamic states generally it is very common to find the bases of the lungs deeply congested, a condition induced partly by the effect of gravity, the patient lying recumbent in one posture for a long time, but chiefly by weakened heart action. That it is not an effect of gravity alone is shown by the fact that a healthy person may remain in bed an indefinite time without its occurrence. The term hypostatic congestion is applied to it. The posterior parts of the lung are dark in color and engorged with blood and serum; in some instances to such a degree that the alveoli no longer contain air and portions of the lung sink in water. The term *splenization* and hypostatic pneumonia have been given to these advanced grades. It is a common affection in protracted cases of typhoid fever and in long debilitating illnesses. In ascites, meteorism, and abdominal tumors the bases of the lungs may be compressed and congested. In this connection must be mentioned the form of passive congestion met with in injury to, and organic disease of, the brain. In cerebral apoplexy the bases of the lungs are deeply engorged, not quite airless, but heavy, and on section drip with blood and serum. I have twice seen this condition in an extreme grade throughout the lungs in death from morphia poisoning. In some instances the lung tissue has a blackish, gelatinous, infiltrated appearance, almost like diffuse pulmonary apoplexy. Occasionally this congestion is most marked in, and even confined to, the hemiplegic side. In prolonged coma the hypostatic congestion may be associated with patches of consolidation, due to the aspiration of portions of food into the air-passages.

The symptoms of hypostatic congestion are not at all characteristic, and the condition has to be sought for by careful examination of the bases of the lungs, when slight dulness, feeble, sometimes blowing, breathing and liquid râles can be detected.

The *treatment* of congestion of the lungs is usually that of the condition with which it is associated. In the intense pulmonary engorgement, which may possibly occur primarily, and which is met with in heart-disease and emphysema, free bleeding should be practised. From twenty to thirty ounces of blood should be taken from the arm, and if the blood does not flow freely and the condition of the patient is desperate, aspiration of the right auricle may be performed.

Œdema.—In all forms of intense congestion of the lungs there is a transudation of serum from the engorged capillaries chiefly into the air-cells, but also into the alveolar walls. Not only is it very frequent in congestion, but also with inflammation, with new growths, infarcts, and tuber-

cles. When limited to the neighborhood of an affected part, the name collateral œdema is sometimes applied to it. General œdema occurs under conditions very similar to those met with in congestion. It is very often, no doubt, a terminal event, occurring with the death agony. It is seen in typical form in the cachexias, in death from anæmia, also in chronic Bright's disease, disease of the heart, and cerebral affections.

The œdematous lung is heavy, looks watery, pits on pressure, and from the cut surface a large quantity of clear and, in cases of congestion, bloody serum flows freely; the tissue may even have a gelatinous, infiltrated appearance. The condition is much more common at the bases, but it may exist throughout the entire lung. The pathology of pulmonary œdema is not always clear. Two factors usually prevail in extreme cases—increased tension within the pulmonary system and a diluted blood plasma. The increased tension alone is not capable of producing it. The experiments of Welch seem to indicate that the essential factor lies in a disproportionate weakness of the left ventricle, so that the blood accumulates in the lung capillaries until transudation occurs, a view which satisfactorily explains certain cases, particularly the terminal œdemas.

The *symptoms* of œdema of the lungs are often only an aggravation of those already existing, and are due to the primary disease, whether cardiac, renal, or general. There are usually increasing dyspnœa and cough, and on examination there may be defective resonance and large liquid râles at the bases. There are cases in which the œdema comes on with great suddenness, and in chronic Bright's disease it may prove rapidly fatal.

In the cases of so-called inflammatory œdema fever is always present, and often signs, more or less marked, of pneumonia.

The *treatment* of œdema of the lung is practically that of the conditions with which it is associated. In the acute cases active catharsis, and, if there is cyanosis, free venesection should be resorted to.

Pulmonary Hæmorrhage.—This occurs in two forms—*broncho-pulmonary hæmorrhage*, sometimes called bronchorrhagia, in which the blood is poured out into the bronchi and is expectorated, and *pulmonary apoplexy* or pneumorrhagia, in which the hæmorrhage takes place into the air-cells and the lung tissue.

1. *Broncho-pulmonary Hæmorrhage; Hæmoptysis.*—Spitting of blood, to which the term hæmoptysis should be restricted, results from a variety of conditions, among which the following are the most important: (*a*) In young healthy persons hæmoptysis may occur without warning, and after continuing for a few days disappear and leave no ill traces. There may be at the time of the attack no physical signs indicating pulmonary disease. In such cases good health may be preserved for years and no further trouble occur. These cases are not very uncommon. In Ware's important contribution to this subject,* of 386 cases of hæmoptysis noted in

* On Hæmoptysis as a Symptom, by John Ware, M. D.

private practice 62 recovered and pulmonary disease did not subsequently develop in them. I know three professional men who had hæmoptysis as students, and who now, at periods of from fifteen to eighteen years subsequently, remain in perfect health. (*b*) Hæmoptysis in pulmonary tuberculosis. So frequently are these conditions associated that in the lay mind spitting of blood and consumption are almost synonymous. The Hippocratic aphorism, "From a spitting of blood there is a spitting of pus," is repeated throughout the literature of more than twenty centuries. It occurs either early in the disease, before there are any obvious physical signs, or after the development of well-marked local lesions. Unquestionably in a majority of the cases in which subsequent to hæmoptysis phthisis occurs tubercles were already present in the lung. The hæmorrhage is bronchial and associated with a limited focus of disease. When the pulmonary lesion is more advanced the hæmoptysis results either from erosion of a branch of the pulmonary artery or from rupture of an aneurismal dilatation of the same. (*c*) In connection with certain diseases of the lung, as pneumonia (in the initial stage) and cancer, occasionally in gangrene, abscess, and bronchiectasis, hæmoptysis occurs. (*d*) Hæmoptysis is met with in many heart affections, particularly mitral lesions. It may be profuse and recur at intervals for years. (*e*) In ulcerative affections of the larynx, trachea, or bronchi. Sometimes the hæmorrhage is profuse and rapidly fatal, as when an ulcer erodes a large branch of the pulmonary artery, an accident which I have known to happen in a case of chronic bronchitis with emphysema. (*f*) Aneurism is an occasional cause of hæmoptysis. It may be sudden and rapidly fatal when the sac bursts into the air-passages. Slight bleeding may continue for weeks or even longer, due to pressure on the mucous membrane, erosion of the lung, or in some cases the sac "weeps" through the exposed laminæ of fibrin. (*g*) Vicarious hæmorrhage, which occurs in rare instances in cases of interrupted menstruation. The instances are well authenticated. Flint mentions a case which he had had under observation for four years, and Hippocrates refers to it in the aphorism, "Hæmoptysis in a woman is removed by an eruption of the menses." Periodical hæmoptysis has also been met with after the removal of both ovaries. Even fatal hæmorrhage has occurred from the lung during menstruation when no lesion was found to account for it. (*h*) There is a form of recurring hæmoptysis in arthritic subjects to which Sir Andrew Clark has called special attention and which also is described by French writers. The cases occur in persons over fifty years of age who usually present signs of the arthritic diathesis. It rarely leads to fatal issue and subsides without inducing pulmonary changes. (*i*) Hæmoptysis recurs sometimes in malignant fevers and in purpura hæmorrhagica. Lastly, there is endemic hæmoptysis, due to the presence of the *Distoma Ringeri* in the bronchial tubes, an affection which is confined to parts of China and Japan.

Symptoms.—Hæmoptysis sets in as a rule suddenly. Often without warning the patient experiences a warm, saltish taste as the mouth

fills with blood. Coughing is usually induced. There may be only an ounce or so brought up before the bleeding stops, or the bleeding may continue for days, the patient bringing up small quantities. In other instances, particularly when a large vessel is eroded or an aneurism bursts, the amount is large, and the patient after a few attempts at coughing shows signs of suffocation and death is produced by inundation of the bronchial system. Fatal hæmorrhage may even occur into a large cavity in a patient debilitated by phthisis without the production of hæmoptysis. I dissected a case of this kind at the Philadelphia Hospital. The blood from the lungs generally has characters which render it readily distinguishable from the blood which is vomited. It is alkaline in reaction, frothy, and mixed with mucus, and when coagulation occurs air-bubbles are present in the clot. Blood-moulds of the smaller bronchi are sometimes seen. Patients can usually tell whether the blood has been brought up by coughing or by vomiting, and in a majority of cases the history gives important indications. In paroxysmal hæmoptysis connected with menstrual disturbances the practitioner should see that the blood is actually coughed up, since deception may be practised. Naturally, the patient is at first alarmed at the occurrence of bleeding, but, unless very profuse, as when due to rupture of an aortic aneurism in a pulmonary cavity, the danger is rarely immediate. The attacks, however, are apt to recur for a few days and the sputa may remain blood-tinged for a longer period. In the great majority of cases the hæmorrhage ceases spontaneously. It should be remembered that some of the blood may be swallowed and produce vomiting, and, after a day or two, the stools may be dark in color. It is not well during an attack of hæmoptysis to examine the chest. It was formerly thought that hæmorrhage exercised a prejudicial effect and excited inflammation of the lungs, but this is not often the case.

(2) *Pulmonary Apoplexy; Hæmorrhagic Infarct.*—In this condition the blood is effused into the air-cells and interstitial tissue. It is rarely indeed diffuse, breaking the parenchyma as the brain tissue is broken in cerebral apoplexy. Sometimes, in disease of the brain, in septic conditions, and in the malignant forms of fevers, the lung tissue is uniformly infiltrated with blood and has, on section, a black, gelatinous appearance.

As a rule, the hæmorrhage is limited and results from the blocking of a branch of the pulmonary artery either by a thrombus or an embolus. The condition is most common in chronic heart-disease. Although the pulmonary arteries are terminal ones, blocking is not always followed by infarction; partly because the wide capillaries furnish sufficient anastomosis, and partly because the bronchial vessels may keep up the circulation. The infarctions are chiefly at the periphery of the lung, usually wedge-shaped, with the base of the wedge toward the surface. When recent, they are dark in color, hard and firm, and look on section like an ordinary blood-clot. Gradual changes go on, and the color becomes a reddish brown. The pleura over an infarct is usually inflamed. A mi-

croscopical section shows the air-cells to be distended with red blood-corpuscles, which may also be in the alveolar walls. The infarcts are usually multiple and vary in size from a walnut to an orange. Very large ones may involve the greater part of a lobe. In the artery passing to the affected territory a thrombus or an embolus is found. The globular thrombi, formed in the right auricular appendix, play an important part in the production of hæmorrhagic infarction. In many cases the source of the embolus cannot be discovered, and the infarct may have resulted from thrombosis in the pulmonary artery, but, as before mentioned, it is not infrequent to find total obstruction of a large branch of a pulmonary artery without hæmorrhage into the corresponding lung area. The further history of an infarction is variable. It is possible that in some instances the circulation is re-established and the blood removed. More commonly, if the patient lives, the usual changes go on in the extravasated blood and ultimately a pigmented, puckered, fibroid patch results. Sloughing may occur with the formation of a cavity. Occasionally gangrene results. In a case at the University Hospital, Philadelphia, a gangrenous infarct ruptured and produced fatal pneumothorax.

The *symptoms* of pulmonary apoplexy are by no means definite. The condition may be suspected in chronic heart-disease when hæmoptysis occurs, particularly in mitral stenosis, but the bleeding may be due to the extreme engorgement. When the infarcts are very large, and particularly in the lower lobe, in which they most commonly occur, there may be signs of consolidation with blowing breathing.

Treatment of Pulmonary Hæmorrhage.—In the treatment of hæmoptysis it is important to remember the condition of the pulmonary circulation and the nature of the lesions associated with the hæmorrhage.

The pressure within the pulmonary artery is considerably less than that in the aortic system. We have as yet very imperfect knowledge of the circumstances which influence the lesser circulation in man. Researches, particularly those of Bradford, indicate that the system is under vasomotor control, but our knowledge of the mutual relations of pressure in the aorta and in the pulmonary artery, under varying conditions, is still very imperfect. Experiments with drugs seem to show that there may be an influence on systemic blood-pressure without any on the pulmonary, and the pressure in the one may rise while it falls in the other, or it may rise and fall in both together. In Andrew's Harveian Oration these relations are thoroughly described, and a statement is made, based on Bradford's experiments, as to the action on the pulmonary blood-pressure of many of the drugs employed in hæmoptysis. Thus ergot, the remedy perhaps most commonly used, causes a distinct rise in the pulmonary blood-pressure, while aconite produces a definite fall.

The anatomical condition in hæmoptysis is either hyperæmia of the bronchial mucosa (or of the lung tissue) or a perforated artery. In the

latter case the patient often passes rapidly beyond treatment, though there are instances of the most profuse hæmorrhage which must have come from a perforated artery or a ruptured aneurism in which recovery has occurred. Practically, for treatment, we should separate these cases, as the remedies which would be applicable in a case of congested and bleeding mucosa would be as much out of place in a case of hæmorrhage from ruptured aneurism as in a cut radial artery. When the blood is brought up in quantities—in mouthfuls at a time—it is almost certain either that an aneurism has ruptured or a vessel has been eroded. In the instances in which the sputa are blood-tinged or when the blood is in smaller quantities, bleeding comes by diapedesis from hyperæmic vessels. In such cases the hæmorrhage may be beneficial in relieving the congested blood-vessels.

The indications are to reduce the frequency of the heart-beats and to lower the blood-pressure. By far the most important measure is absolute quiet of body, such as can only be secured by rest in bed and seclusion. In the majority of cases of mild hæmoptysis this is sufficient. Even when the patient insists upon going about, the bleeding may stop spontaneously. The diet should be light and unstimulating. Alcohol should not be used. The patient may, if he wishes, have ice to suck. Small doses of aromatic sulphuric acid may be given, but unless the bleeding is protracted styptic and astringent medicines are not indicated. For cough, which is always present and disturbing, opium should be freely given, and is of all medicines most serviceable in hæmoptysis. Digitalis should not be used, as it raises the blood-pressure in the pulmonary artery. Aconite, as it lowers the pressure, may be used when there is much vascular excitement. Ergot, tannic acid, and lead, which are so much employed, have, I believe, little or no influence in hæmoptysis. Ergot, according to Bradford, produces distinct rise in the pulmonary blood-pressure. One of the most satisfactory means of lowering the blood-pressure is purgation, and when the bleeding is protracted salts may be freely given. In profuse hæmoptysis, such as comes from erosion of an artery or the rupture of an aneurism, a fatal result is common, and yet post-mortem evidence shows that thrombosis may occur with healing in a rupture of considerable size. The fainting induced by the loss of blood is probably the most efficient means of promoting thrombosis, and it was on this principle that formerly patients were bled from the arm, or from both arms, as in the case of Laurence Sterne. Ligatures, or Esmarch's bandages, placed around the legs may serve temporarily to check the bleeding. The ice-bag on the sternum is of doubtful utility. In a protracted case Cayley induced pneumothorax, but without effect.

Briefly, then, we may say that cases of hæmorrhage from rupture of aneurism or erosion of a blood-vessel usually prove fatal. The fainting induced by the loss of blood is beneficial, and, if the patient can be kept alive for twenty-four hours, a thrombus of sufficient strength to prevent further bleeding may form. The chief danger is the inundation of the

bronchial system with the blood, so that while the hæmorrhage is profuse the cough should be encouraged. Opium should not then be used, and stimulants should be given with caution.

In the other group, in which the hæmorrhage comes from a congested area and is limited, the patient gets well if kept absolutely quiet, and fatal hæmorrhage probably never occurs from this source. Rest, reduction of the blood-pressure by minimum diet, purging, if necessary, and the administration of opium to allay the cough are the main indications.

II. PNEUMONIA

(Lobar, Croupous, or Fibrinous Pneumonia; Pneumonitis; Lung Fever).

Definition.—An infectious disease characterized by inflammation of the lungs and constitutional disturbance of varying intensity. The fever terminates abruptly by crisis. Secondary infective processes are common An organism, the *diplococcus pneumoniæ*, is invariably found in the diseased lung.

Etiology.—Pneumonia is one of the most wide-spread of acute diseases. Hospital statistics show that the ratio to other admissions is in the proportion of twenty to thirty per thousand.

It prevails at all ages Children are quite as susceptible to it as adults, and it is the special enemy of old age. Males are more frequently affected than females. Dwellers in cities and persons whose occupations are associated with exposure, hardship, and cold are most liable to the disease. Contrary to the general rule in infectious diseases, newcomers and immigrants seem less susceptible than the native inhabitants. Debilitating causes of all sorts render individuals more susceptible. Alcoholism is perhaps the most potent predisposing factor. Persons weakened by disease are especially prone to it; thus we find many cases in connection with chronic Bright's disease, diabetes, the chronic affections of the nervous system, and protracted fevers. One important predisposing cause is a previous attack. No acute disease recurs with such frequency. Instances are on record of individuals who have had ten or more attacks.

Climate does not appear to have much influence. The disease prevails equally in cold and in hot countries, but it is stated that on this continent it is more prevalent in the Southern than in the Northern States. More important is the influence of season. Statistics everywhere show that more persons are attacked from December to May than in the summer and autumn. Seitz's large statistics of 5,905 cases in Munich give 32 per cent in winter, 36·8 per cent in spring, 15·3 per cent in summer, and 15·7 per cent in autumn. Bell's statistics of the Montreal General Hospital show practically the same distribution, but it is worth noting that during January, the coldest month of the year, in which the mean temperature for ten years was 13·75° F., the percentage was compara-

tively low. January, however, is a month with very slight variations in temperature, and it seems that the sudden changes characteristic of March, April, and May are the important climatic factors which predispose to pneumonia.

Of other factors, cold has been thought to be one of the most important, and for years was regarded as the efficient cause of the disease. Undoubtedly the disease sometimes promptly follows a sudden chilling or wetting, but in a large majority of cases no such history can be obtained.

Pneumonia follows traumatism with great frequency, more particularly injury of the chest. Litten has called special attention to this so-called *contusions-pneumonia*.

A change of opinion has of late taken place as to the nature of pneumonia, which is now almost universally regarded as a specific infectious disease, depending upon a micro-organism. Among general circumstances favoring this view, is the occurrence of pneumonia in *epidemic form*, a fact recognized by Laennec and by Grisolle. Many house epidemics have been described within the past twenty years. On several occasions I have known two, three, and even four persons admitted to hospital from the same house. In 1887, I saw, with Graham, of Toronto, a local outbreak in which three members of a family were consecutively attacked with the most malignant pneumonia. There are instances on record in which as many as ten residents in one house have been attacked. Of late years many epidemics in towns have been reported. Still more striking are the epidemics which have been described in prisons and garrisons, of which one of the most remarkable is that reported by W. B. Rodman, of Frankfort, Kentucky. In one year there occurred in a prison population of 735, 118 cases, with 25 deaths. The prison was much overcrowded at the time. Similar epidemics have been described in Europe. At the penitentiary at Amberg, from the 1st of January to the 1st of June, there were 161 cases of pneumonia with a mortality of over twenty-eight per cent.

The *diplococcus pneumoniæ* of Fraenkel is the most constant organism in lobar pneumonia and is now believed by many competent authorities to be the specific agent of the disease. It is identical with the micrococcus which Pasteur and Sternberg found in the saliva of certain individuals and which produces septicæmia in the rabbit. It occurs occasionally in the nose, the larynx, and the Eustachian tube. According to Netter's observations, it is present in the buccal secretion in twenty per cent of healthy persons. It persists for months or even years in the saliva of persons who have had pneumonia. The researches of Fraenkel, Weichselbaum, Gamaleia, and others show that it is by far the most constant organism in pneumonia and that it occurs in the secondary processes of the disease, such as pleurisy, endocarditis, pericarditis, and meningitis. In ten cases recently examined at the pathological laboratory of the Johns Hopkins Hospital by my colleague Welch, this organism was present in all; in

six as pure cultures in the lung, in four together with pus organisms. In the sputum it may be demonstrated by treating the ordinary cover-glass preparations with glacial acetic acid and then, without washing off the acid, dropping on aniline oil and gentian-violet, which is to be poured off and renewed two or three times. The organism is seen to be a somewhat elliptical lance-shaped coccus occurring in pairs, hence the term diplococcus. It is usually encapsulated.

According to the dominant view, pneumonia is an infective disease caused by this diplococcus, which has its seat of election in and produces its chief effects on the lung, and which can, under favoring circumstances, invade other parts of the body—the pleura, meninges, and endocardium This microbe may possibly attack these parts without the intervention of inflammation of the lung, as it has been found in meningitis and pleurisy independent of pneumonia. It is a wide-spread organism, at times present, as before stated, in the buccal secretions of healthy persons. It is not improbable that the various predisposing causes, such as cold, exhaustion, and debility, lower the vitality and render the individual susceptible, thus changing the character of the tissue-soil so that the virus can grow and produce its specific effects.

On this view, pneumonia may be regarded as a local disease, produced by micro-organisms which induce, as in other local diseases, such as erysipelas and diphtheria, constitutional disturbance of varying degrees of intensity, or even, by the further invasion of the parasites, secondary infective processes in other organs.*

Recently from Leyden's clinic very interesting studies have been issued by the brothers Klemperer on the production of immunity and upon the cure of pneumonia. Immunity is readily obtained in animals either by subcutaneous or intravenous injections of large quantities of the filtered bouillon cultures, or by the injection of the glycerine extract. The immunity, though rarely lasting more than six months, was transmitted to the offspring born within this period. Still more interesting are their observations upon the cure of the experimentally produced disease. They found that the serum and fluids of the body of an animal which had been rendered immune had the property not only of producing immunity when introduced into the circulation of another susceptible animal, but actually of curing the disease after infection had been in progress for some time. In infected animals with a body temperature of from 40° to 41° C., the fever fell to normal in twenty-four hours after the injection of serum of another animal which possessed immunity. They believe that the pneumococcus produces a poisonous albumen (pneumotoxin) which when introduced into the circulation of an animal causes elevation of temperature and the subsequent production in the body of a substance

* See on the question of etiology the elaborate essay of Wells, Journal of the American Medical Association, 1889.

(antipneumotoxin) which possesses the power of neutralizing the poisonous albumen which is formed by the bacteria. In man they hold that during the pneumonic process there is a constant absorption into the circulation of this poisonous albumen produced by the bacteria in the lungs. This continues until eventually the same antidotal substance is produced in the circulation that has been seen to occur experimentally. It is then that the crisis occurs. The bacteria are neither destroyed nor is their power to produce the poisonous albumen lessened, but the third factor, the antitoxic element, now exists and neutralizes the toxic substances as they are produced. They demonstrated that the serum of the blood of patients after the crisis of pneumonia contained the antitoxic substance and was capable, in a fair number of cases, of curing the disease when injected into infected animals. They have made preliminary observations upon patients with a view of inducing the crisis by the injection of the blood serum of persons convalescent from pneumonia, and which consequently contains the antitoxic body. In six pneumonic patients the results were promising. In all there was a decided fall of temperature in from six to twelve hours after subcutaneous injections of from four to six c. c. of the serum. The pulse and respirations were also diminished in frequency. In two cases the temperature fell to 37° C. Twice it fell and remained at normal. In the other cases it fell only temporarily. In two typhoid cases the injections were negative. The serum has no effect when injected into healthy individuals.

Morbid Anatomy.—Since the time of Laennec, pathologists have recognized three stages in the inflamed lung—engorgement, red hepatization, and gray hepatization.

In the stage of *engorgement* the lung tissue is deep red in color, firmer to the touch, and more solid, and on section the surface is bathed with blood and serum. It still crepitates, though not so distinctly as healthy lung, and excised portions float. The air-cells can be dilated by insufflation from the bronchus. Microscopical examination shows the capillary vessels to be greatly distended, the alveolar epithelium swollen, and the air-cells occupied by a variable number of blood-corpuscles and detached alveolar cells. In the stage of *red hepatization* the lung tissue is solid, firm, and airless. If the entire lobe is involved it looks voluminous, and shows indentations of the ribs. On section the surface is dry, reddish brown in color, and has lost the deeply congested appearance of the first stage. One of the most remarkable features is the friability; in striking contrast to the healthy lung, which is torn with difficulty, a hepatized organ can be readily broken by the finger. Careful inspection shows that the surface is distinctly granular, the granulations representing fibrinous plugs filling the air-cells. The distinctness of this appearance varies greatly with the size of the alveoli, which are about 0·10 mm. in diameter in the infant, 0·15 or 0·16 in the adult, and from 0·20 to 0·25 in old age. On scraping the surface with a knife a reddish viscid serum is

removed, containing small granular masses. The smaller bronchi often contain fibrinous plugs. If the lung has been removed before the heart, it is not uncommon to find solid moulds of clot filling the blood-vessels. Microscopically, the air-cells are seen to be occupied by coagulated fibrin in the meshes of which are red blood-corpuscles, polynuclear leucocytes, and alveolar epithelium. The alveolar walls are infiltrated and leucocytes are seen in the interlobular tissues. Cover-glass preparations from the exudate, and thin sections show, as a rule, the diplococci already referred to, many of which are contained within cells. Staphylococci and streptococci may also be seen in some cases. In the stage of *gray hepatization* the tissue has changed from a reddish-brown to a grayish-white color. The surface is moister, the exudate obtained on scraping is more turbid, the granules in the acini are less distinct, and the lung tissue is still more friable. Histologically, in gray hepatization, it is seen that the air-cells are densely filled with leucocytes, the fibrin network and the red blood-corpuscles have disappeared. A more advanced condition of gray hepatization is that known as *purulent infiltration*, in which the lung tissue is softer and bathed with a purulent fluid.

The stage of gray hepatization appears to be the first step in the process of *resolution*. The exudate is softened, the cell elements are disintegrated and rendered capable of absorption. When the purulent infiltration of the lung tissue reaches the grade sometimes seen post mortem, it is probable that resolution could not take place. Small abscess cavities may arise, and by their fusion larger ones. Often in one lung, or even in one lobe, the various stages of the process may be seen, and the passage of the engorgement into red hepatization and of the latter into the gray stage can be readily traced.

The general details of the morbid anatomy of pneumonia may be gathered from the following facts, based on 100 autopsies, made by me at the General Hospital, Montreal: In 51 cases the right lung was affected; in 32, the left; in 17, both organs. In 27 cases the entire lung, with the exception, perhaps, of a narrow margin at the apex and anterior border, was consolidated. In 34 cases, the lower lobe alone was involved; in 13 cases, the upper lobe alone. When double, the lower lobes were usually affected together, but in three instances the lower lobe of one and the upper lobe of the other were attacked. In three cases also, both upper lobes were affected. Occasionally the disease involves the greater part of both lungs; thus, in one instance the left organ with the exception of the anterior border was uniformly hepatized, while the right was in a stage of gray hepatization, except a still smaller portion in the corresponding region. In a third of the cases, red and gray hepatization existed together. In 22 instances there was gray hepatization. As a rule the unaffected portion of the lung is usually congested or œdematous. When the greater portion of a lobe is attacked, the uninvolved part may be in a state of almost gelatinous œdema. The unaffected lung is usually congested, particularly

at the posterior part. This, it must be remembered, may be largely due to post-mortem subsidence. The uninflamed portions are not always congested and œdematous. The upper lobe may be dry and bloodless when the lower lobe is uniformly consolidated. The average weight of a normal lung is about 600 grammes, while that of an inflamed organ may be 1,500, 2,000, or even 2,500 grammes.

The bronchi contain, as a rule, at the time of death a frothy serous fluid, rarely the tenacious mucus so characteristic of pneumonic sputum. The mucous membrane is usually reddened, rarely swollen. In the affected areas the smaller bronchi often contain fibrinous plugs, which may extend into the larger tubes, forming perfect casts. The bronchial glands are swollen and may even be soft and pulpy. The pleural surface of the inflamed lung is invariably involved when the process becomes superficial. Commonly, there is only a thin sheeting of exudate, producing slight turbidity of the membrane. In only two of the hundred instances the pleura was not involved. In some cases the fibrinous exudate may form a creamy layer an inch in thickness. A serous exudation of variable amount is not uncommon.

Lesions in other Organs.—The heart is distended with firm, tenacious coagula, which can be withdrawn from the vessels as dendritic moulds. In no other acute disease do we meet with coagula of such solidity and firmness. The distention of the right chambers of the heart is particularly marked. The left chambers are rarely distended to the same degree. The spleen is often enlarged, though in only 35 of the 100 cases was the weight above 200 grammes. The kidneys show parenchymatous swelling, turbidity of the cortex, and, in a very considerable proportion of the cases —twenty-five per cent—chronic interstitial changes.

Pericarditis is not infrequent, and occurs more particularly with pneumonia of the left side and with double pneumonia. In 5 of the 100 autopsies it was present, and in 4 of them the lappet of lung overlying the pericardium with its pleura was involved. Endocarditis is more frequent and occurred in 16 of the 100 cases. In 5 of these the endocarditis was of the simple character; in 11 the lesions were ulcerative. Fatty degeneration of the heart is not common except in protracted cases.

Meningitis is not infrequently found, and in many cases is associated with malignant endocarditis. It was present in 8 of the 100 autopsies. Of twenty cases of meningitis in ulcerative endocarditis fifteen occurred in pneumonia. The meningeal inflammation in these cases is usually cortical.

Croupous or diphtheritic inflammation may occur in other parts. A croupous colitis, as pointed out by Bristowe, is not very uncommon. It occurred in 5 of my 100 post-mortems. It is usually a thin, flaky exudation, most marked on the tops of the folds of the mucous membrane. In one case there was a patch of croupous gastritis, covering an area of 12 by 8 cm., situated to the left of the cardiac orifice.

The liver shows parenchymatous changes and often extreme engorgement of the hepatic veins.

Symptoms.—Abruptly, or preceded by a day or two of indisposition, the patient has a severe chill, lasting from ten to thirty minutes. In no acute disease is an initial chill so constant or so severe. The fever rises quickly. There is pain in the side, often of an agonizing character. A short, dry, painful cough soon develops, and the respirations are increased in frequency. When seen on the second or third day the patient presents an appearance which may be quite pathognomonic. He lies flat in bed, often on the affected side; the face is flushed, particularly the cheeks; the breathing is hurried; the alæ nasi dilate with each inspiration; the eyes are bright, the expression is anxious, and there is a frequent short cough which makes the patient wince and hold his side. The expectoration is blood-tinged and extremely tenacious. The temperature rises rapidly to 104° or 105°. The pulse is full and bounding and the pulse-respiration ratio much disturbed. Examination of the lung shows the physical signs of consolidation—blowing breathing and fine râles. After persisting for from seven to ten days the crisis occurs, and with a fall in the temperature the patient passes from a condition of extreme distress and anxiety to one of comparative comfort.

The fever of pneumonia rises abruptly with the chill, during which the rectal temperature may be high. In children and in cases without chill the rise is more gradual. The temperature reaches 104° or 105° and is continuous, with a variation of a degree to a degree and a half. If a two-hour record is kept the diurnal variations are seen to follow the normal type. In children and healthy adults the fever is usually higher than in old persons and drunkards. After continuing for from five to nine days the temperature falls abruptly, forming what is known as the *crisis*, so characteristic in a large proportion of the cases. In from five to twelve hours the temperature may fall eight degrees. The crisis may occur as early as the third day or as late as the twelfth or fourteenth. A *pseudo-crisis* may occur on the fifth day or earlier. Defervescence may take place gradually by lysis. In cases of delayed resolution the fever may persist for weeks.

Respiratory Symptoms.—Pain of an agonizing character is an early and distressing symptom. It is usually referred to the nipple or axillary regions of the affected side. In exceptional cases it may be in the abdomen or flank, or even beneath the shoulder-blade. Deep inspiration and cough aggravate it. Dyspnœa is a very prominent feature. The respirations may be from forty to sixty in the minute and in exceptional cases and in children may rise to eighty. To produce this shortness of breath many factors combine—the fever, the loss of function in a considerable area of lung tissue, and the excessive pain in the side, which makes it impossible to draw a deep breath. There may be nervous factors at work, as with the crisis the number of respirations may fall nearly to normal,

while the consolidation of the lung still persists. The type of breathing in pneumonia is peculiar and almost distinctive. The inspirations are

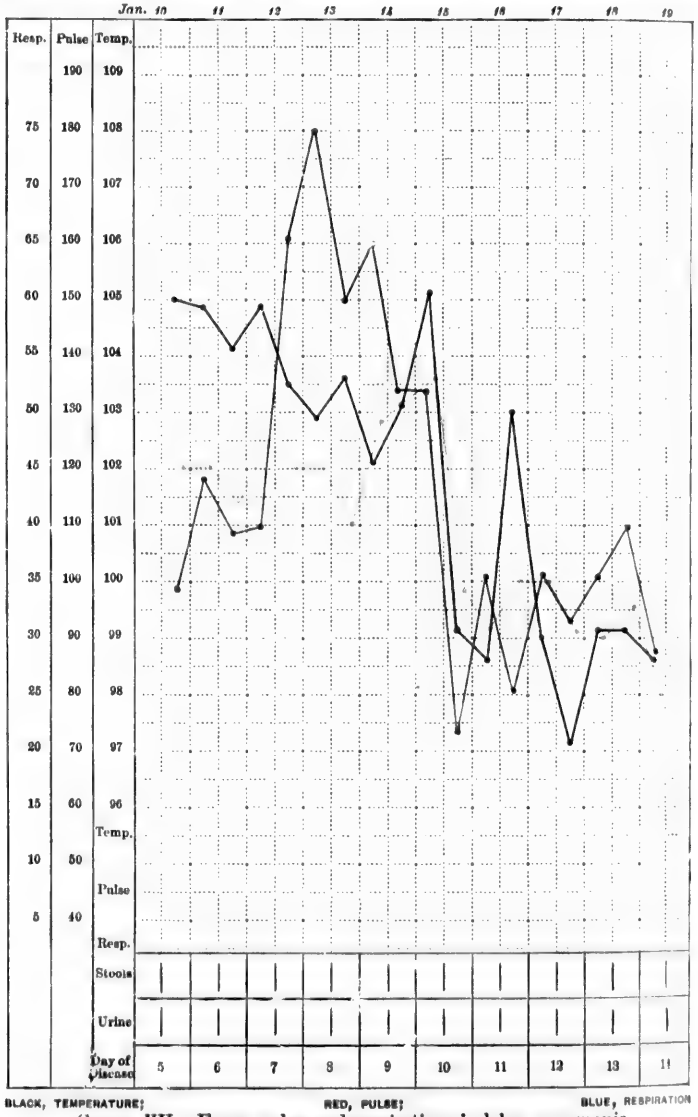


CHART XV.—Fever, pulse, and respirations in lobar pneumonia.

short and superficial. Expiration is often associated with a short grunt. The ratio between the respirations and pulse may be 1 to 2, or even 1 to

1·5. In no other disease do we see such marked disturbance in the pulse-respiration ratio, and this is sometimes an aid in diagnosis.

The cough is also very characteristic—frequent, short, restrained, and associated with great pain in the side. It is at first dry, hard, and without expectoration. In old persons and drunkards and in those debilitated by long illness there may be no cough. The sputum is mucoid at first, but within twenty-four hours shows special features. A brisk hæmoptysis may be an initial symptom. Pneumonic sputum is viscid, tenacious, and blood-tinged. The gummous viscidity, together with the red blood-corpuscles in various stages of alteration, give pathognomonic characters to the sputa, unknown in any other disease. The rusty tinge becomes more marked as the disease progresses, and so tenacious is the expectoration that it has to be wiped from the lips of the patient, and a spit-cup, half full, may be inverted without spilling. Toward the close it becomes more liquid and is more readily expelled. In low types of the disease the sputum may be fluid and dark brown, resembling prune juice. The amount is very variable. In children and old people there may be none; ordinarily, however, there are from 100 to 300 c. c. daily. After the crisis the quantity is variable; abundant in some cases, absent in others. Microscopically, the sputum contains red blood-corpuscles in all stages of degeneration, alveolar epithelium, diplococci and other micro-organisms, cell-moulds of the alveoli, and, in some cases, small fibrinous casts of the bronchioles. The latter are sometimes plainly visible to the naked eye.

Physical Signs.—*Inspection* may not at first show any difference between the two sides, though usually if the lower lobe of a lung is involved the movement is less on the affected side. Later, when consolidation has occurred, particularly if it is massive, this deficient expansion is very marked. Mensuration may show a definite increase in the volume of the side involved. The intercostal spaces are not obliterated. Palpation indicates still more clearly the lack of expansion, and a pleural friction may be felt. Tactile fremitus is increased. These signs are all more marked when consolidation is established.

Percussion.—In the stage of engorgement the note is higher pitched and may have a somewhat tympanitic quality, the so-called Skoda's resonance. This can often be obtained over the lung tissue just above a consolidated area. When the lung is hepatized, the percussion note is flat, the quality of the flatness varying a good deal from a note which has in it a certain tympanitic quality to absolute dulness. There is not the wooden flatness of effusion and the sense of resistance is not so great. During resolution the tympanitic quality of the percussion note may return. For weeks or months after convalescence there may be a higher-pitched note on the affected side.

Auscultation.—Quiet, suppressed breathing in the affected part is often a marked feature in the early stage, and is always suggestive. Very early there is heard at the end of inspiration the fine crepitant râle, a series of

minute cracklings heard close to the ear, and perhaps not audible until a full breath is drawn. Whether this is a fine pleural crepitus or is produced in the air-cells and finer bronchi is still an open question. At this stage, before consolidation has occurred, the breath-sounds may be, as before mentioned, much feebler than in health, but on drawing a long breath they may have a harsh quality, to which the term broncho-vesicular has been applied. In the stage of red hepatization and when dulness is well defined, the respiration is tubular, similar to that heard in health over the larger bronchi. With this blowing breathing there may be no râles, and it may present an intensity unknown in any other pulmonary affection. It is simply the propagation of the laryngeal and tracheal sounds through the bronchi and the consolidated lung tissue. The permeability of the bronchi is essential to its production. Tubular breathing is absent in certain cases of massive pneumonia in which the larger bronchi are completely filled with exudation. When resolution begins mucous râles of all sizes can be heard. At first they are small and have been called the *redux-crepitus*. The voice-sounds are transmitted through the consolidated lung with great intensity. This bronchophony may have a curious nasal quality to which the term ægophony has been given.

Circulatory Symptoms.—During the chill the pulse is small, but in the succeeding fever it becomes full and bounding. In cases of moderate severity it ranges from 100 to 116. It is not often dicrotic. In strong, healthy individuals and in children there may be no sign of failing pulse throughout the attack. With extensive consolidation the left ventricle may receive a very diminished amount of blood and the pulse in consequence may be small.

In the old and feeble the pulse may be small and rapid from the outset. The heart-sounds are usually loud and clear. During the intensity of the fever, particularly in children, *bruits* are not uncommon both in the mitral and in the pulmonary areas. The second sound over the pulmonary artery is accentuated. Attention to this sign gives a valuable indication as to the condition of the lesser circulation. With distention of the right chambers and failure of the right ventricle to empty itself completely the pulmonary second sound becomes much less distinct. When the right heart is engorged there may be an increase in the dulness to the right of the sternum. With gradual heart-failure and signs of dilatation the long pause is greatly shortened, the sounds approach each other in tone and have a fœtal character (embryocardia).

Blood.—The number of red corpuscles is reduced, but, in consequence of the comparative shortness of the attack, we rarely see the anæmia associated with other febrile disorders. No special changes occur in the corpuscles themselves. The colorless corpuscles are increased in number from about 6,000 per c. mm., the normal number, to 10,000, or even more. This leucocytosis disappears as soon as crisis occurs. Its absence during the fastigium is considered to indicate an unfavorable prognosis. A striking

feature in the blood-slide is the richness and density of the fibrin network. This corresponds to the great increase in the fibrin elements, which has long been known to occur in pneumonia, the proportion rising from four to ten parts per thousand. Hayem describes the blood-plates as greatly increased. The diplococci can very rarely be demonstrated in the blood.

The *gastro-intestinal symptoms* are those associated with an ordinary sthenic fever. Vomiting is not frequent at the outset. There is naturally loss of appetite. The tongue is white and furred, and, in cases of a low type, rapidly becomes dry. Constipation is more common than diarrhœa, which does prevail, however, in some epidemics. The spleen is usually enlarged, and the edge can be felt during a deep inspiration. Except in cases of extreme engorgement of the right heart, the liver is usually not increased in volume.

Among *cutaneous* symptoms one of the most interesting is the association of herpes with pneumonia. Not excepting malaria, we see labial herpes more frequently in this than in any other disease, occurring, as it does, in from twelve to forty per cent of the cases. It is supposed to be of favorable prognosis, and figures have been quoted in proof of this assertion. It may also occur on the nose or on the genitals. Its significance and relation to the disease are unknown. It is scarcely necessary to mention the theory which has been advanced, that it is an external expression of a neuritis which involves the pneumogastric and induces the pneumonia. At the height of the disease sweats are not common, but at the crisis they may be profuse. Redness of one cheek is a phenomenon long recognized in connection with pneumonia, and is usually on the same side as the disease.

The *urine* presents the usual febrile characters of high color, high specific gravity, high density, and increased acidity. The nitrogenous elements, urea and uric acid, are notably increased. The chlorides are absent, or greatly reduced, during the height of the fever—due, it is supposed, to the amount exuded in the hepatized lung. At the crisis there may be marked increase in the amount of urine, which is heavily laden with urates and extractives. When jaundice occurs there is bile-pigment. A trace of albumen is present in a large proportion of the cases. It is rarely of serious significance, and seldom associated with tube-casts.

Cerebral Symptoms.—As an initial symptom, headache is common. Consciousness is usually retained throughout, even in severe cases. In children convulsions are common, and in at least one half the cases usher in the disease. There may be violent maniacal symptoms in the adult. I once performed an autopsy in a case of this kind in which there was no suspicion whatever that the disease was other than acute mania. In drunkards the symptoms from the outset may be those of delirium tremens, in which disease it should be an invariable rule, even if fever is not present, to examine the lungs. These patients are apt to wander about, and must

be carefully watched. The preliminary excitement and delirium may give place to hebetude, which deepens to coma. It is not possible to decide in these cases whether meningitis is present or not, since it is usually cortical, and there are no symptoms of pressure on the nerves. In only one of eight instances was there involvement of the base, rendering clear the diagnosis of meningitis. These cases of so-called cerebral pneumonia are frequently associated with very high fever. In senile and alcoholic pneumonia, however, the temperature may be low and yet brain symptoms very pronounced. Mental disturbance may persist during and after convalescence, and insanity develops in a few cases. It is currently stated that apex pneumonia is more often complicated with severe delirium, but it has not been so in my experience.

Complications.—Many of these seem to depend directly on the invasion of the body by the diplococci.

As already mentioned, *pleurisy* is an inevitable event when the inflammation reaches the surface of the lung, and thus can scarcely be termed a complication. But there are cases in which the pleuritic features take the first place—cases to which the term pleuro-pneumonia is applicable. The exudation may be sero-fibrinous with copious effusion, differing from that of an ordinary acute pleurisy in the greater richness of the fibrin, which may form thick, tenacious, curdy layers. Pneumonia on one side with extensive pleurisy on the other is sometimes a puzzling complication to diagnose and an aspirator needle may be required to settle the question. The bacteriological examination of the fluid has demonstrated, in a large number of cases, the presence of the pneumococcus. Of late, special attention has been paid to the frequency with which empyema complicates pneumonia. Effusion may not have been suspected during the height of the disease, but after the temperature has been normal for some days a slight rise occurs and the irregular fever persists. Dulness continues at the base, or may have extended. The breathing is feeble and there are no râles. Such a condition may be closely simulated, of course, by the thickened pleural layers which are so commonly found after the pneumonia. The question should be settled at once by the introduction of the needle. It is by no means an uncommon complication, and many cases of empyema supposed to be primary are in reality secondary to a slight pneumonia.

Pericarditis is more common in the pneumonia of children, particularly when double, and it is said with the pneumonia of the left side. It was present, as I stated, in five of my one hundred autopsies. Though usually plastic, there may be much serous effusion. There is rarely any difficulty in the diagnosis, but when the pneumonia involves the portion of lung covering the pericardium, there may be difficulty in determining, by physical signs, the existence of fluid. The increase in the dyspnœa, the greater feebleness of the pulse, and the gradual suppression of the heart-sounds will give the most valuable indications. In some instances

the fluid is purulent. Though a very serious event, it is surprising how often recovery takes place even in the most desperate cases of pneumonia complicated with pericarditis. I remember that the late Dr. Murchison some years ago commented upon this feature in a case at St. Thomas's Hospital.

Endocarditis is still more frequent, and in my one hundred autopsies was present in sixteen. I called attention in the Gulstonian lectures for 1885 to the great frequency of this complication. Of 209 cases of malignant endocarditis collected from the literature, 54 cases occurred in this disease. Subsequent observations have fully confirmed this statement. It may be said that with no acute febrile disease is endocarditis so frequently associated. It is much more common in the left heart than in the right. It is particularly liable to attack persons with old valvular disease. There may be no symptoms indicative of this complication even in very severe cases. It may, however, be suspected in cases (1) in which the fever is protracted and irregular; (2) when signs of septic mischief arise, such as chills and sweats; (3) when embolic phenomena appear. The frequent complication of meningitis with the endocarditis of pneumonia, which has already been mentioned, gives prominence to the cerebral symptoms in these cases. The physical signs may be very deceptive. There are instances in which no cardiac murmurs have been heard. In others the development under observation of a loud, rough murmur, particularly if diastolic, is extremely suggestive.

Changes in the myocardium are not uncommon, rarely more, however, than cloudy swelling of the fibres; but in some instances there is fatty change.

Ante-mortem heart-clots are rare in pneumonia, even in the extreme grade of dilatation of the right chamber. In not a single instance of my autopsies were there globular thrombi in the auricles or in apices of the ventricles. In protracted cases thrombi occasionally form in the veins. A rare complication is embolism of one of the larger arteries. I saw an instance in Montreal of embolism of the femoral artery at the height of pneumonia, which necessitated amputation at the thigh. The patient recovered.

By far the most important complication is the pneumonic *meningitis*, which varies much at different times and in different places. My Montreal experience is rather exceptional, as eight per cent of the fatal cases had this complication. It usually comes on at the height of the fever and in the majority of the cases is not recognized unless, as before mentioned, the base is involved, which is not common. Meningitis may develop later in the disease and is then more easily diagnosed. Associated as it so often is with ulcerative endocarditis, there may be embolism of the cerebral arteries, inducing hemiplegia. Among rare complications may be mentioned *peripheral neuritis*, of which several instances have been described. I saw one well-marked instance, following pneumonia and influenza, in the

spring of 1890. There was neuritis of the left arm with considerable wasting.

Serious *gastric complications* are rare. A croupous gastritis has already been mentioned. The *croupous colitis* may induce severe diarrhœa. *Jaundice* is one of the most interesting complications of pneumonia and occurs with curious irregularity in different outbreaks of the disease. It sets in early, is rarely very intense, and has not the characters of obstructive jaundice. There are cases in which it assumes a very serious form. The mode of production is not well ascertained. It does not appear to bear any definite relation to the degree of hepatic engorgement and it is certainly not due to catarrh of the ducts. Possibly it may be, in great part, hæmatogenous.

Parotitis occasionally occurs, commonly in association with endocarditis.

A rare complication of pneumonia is an *arthritis* resembling rheumatism, which may come on gradually during the disease or in the convalescence.

Bright's disease does not often follow pneumonia. Peritonitis is exceedingly rare.

Relapse in pneumonia is so uncommon that some good observers have doubted its occurrence. I have never seen an instance in which I was certain that there was a definite relapse. There are cases in which from the ninth to the eleventh day the fever subsides, and after the temperature has been normal for a day or two, a rise occurs and fever may persist for another ten days or even two weeks. Though this might be termed a relapse, it is more correct to regard it as an instance of an anomalous course of delayed resolution. Wagner, who has studied the subject carefully, says that in his large experience of 1,100 cases he met with only 3 doubtful cases. When it does occur, the attack is usually abortive and mild.

Recurrence is more common in pneumonia than in any other acute disease. Rush gives an instance in which there were twenty-eight attacks. Other authorities narrate cases of eight, ten, and even more attacks.

Formerly it was much disputed whether ordinary lobar pneumonia ever terminated in pulmonary phthisis. These are really cases of tuberculo-pneumonic phthisis the onset of which may resemble acute pneumonia.

Clinical Varieties.—A number of different forms of pneumonia have been recognized, such as malignant, adynamic, bilious, malarial, rheumatic, and the like, but they scarcely require a full description. A malarial pneumonia is described and is thought to be very prevalent in some parts of this country. Although I have seen during the past seven years several hundred cases of malaria and am familiar with the bronchial trouble so commonly associated with it, I have yet to see an instance of pneumonia which seemed in any way connected with paludism. The so-called

rheumatic pneumonia has, so far as I know, no peculiarities; nor has rheumatism, I think, any special relation to the disease. The term typhoid pneumonia is commonly used to designate cases with adynamic symptoms and it is to be distinguished from those cases in which typhoid fever begins with a definite pneumonia, the so-called *pneumo-typhus* of foreign writers.

Epidemic pneumonia is, as a rule, more fatal and may display minor peculiarities which differ in different epidemics. In some the cerebral complications are marked; in others, the cardiac. There may be diarrhœa. The pneumonia which occurs with influenza, and was so common in the last epidemic, presents no special features other than the peculiarities of onset. Perhaps, also, it was more severe and more fatal. In diabetic patients pneumonia runs a rapid and severe course, ending sometimes in abscess or gangrene. In the subjects of chronic alcoholism the onset of pneumonia is insidious, the symptoms may be masked, the fever slight, and the clinical picture may be that of delirium tremens. So latent is the disease in some of these cases that the thermometer alone may indicate the presence of an acute disease.

At the extremes of life pneumonia presents certain well-marked features. It is sometimes seen in the new-born. In *infants* it very often sets in with a convulsion. The summit of the lung seems more frequently involved than in adults and the cerebral symptoms are more marked throughout. The torpor and coma, particularly if they follow convulsions, and the preliminary stage of excitement, may lead to the diagnosis of meningitis. Holt has recently published figures which indicate that lobar pneumonia is not uncommon in infants under two years of age. Pneumonic sputum is rarely seen in children.

In *old age* pneumonia may be latent, coming on without chill; the cough and expectoration are slight, the physical signs ill-defined and changeable, and the constitutional symptoms out of all proportion to the extent of the local lesion.

When pneumonia is prevailing extensively, particularly in jails and garrisons, cases are found which have some of the initial symptoms of the disease—perhaps a slight chill, moderate fever, and a few indefinite local signs. This is the so-called *larval pneumonia*. *Apex pneumonia* is said to be more often associated with adynamic features and with marked cerebral symptoms. The expectoration and cough may be slight. I cannot say that in my experience the cerebral symptoms in adults have been more marked in this form, nor do I think it necessarily graver than if situated at the base.

The creeping or *migratory pneumonia* successively involves one lobe after the other and is a peculiar and well-recognized variety.

Double pneumonia presents no peculiarities other than the greater danger connected with it. The term *massive pneumonia* is applied to the rare condition in which not alone the air-cells but the bronchi of the entire

lobe or even of the lung are filled with the fibrinous exudate. The auscultatory signs are absent; there is neither fremitus nor tubular breathing, and on percussion the lung is absolutely flat. It closely resembles pleurisy with effusion. The moulds of the bronchi may be expectorated in violent fits of coughing.

Prognosis.—In a disease which carries off one in every four or five of those attacked the prognosis in a large number of cases is necessarily grave. In children and in healthy adults the outlook is good. In the debilitated, in drunkards, and in the aged the chances are against recovery. So fatal is it in the latter class that it has been termed the natural end of the old man. Many circumstances, of course, influence prognosis, particularly the extent of the disease, the height of the fever, the presence of other diseases, and the occurrence of complications.

When a lower lobe on one side or the lower and middle lobes of the right side are involved in a healthy adult, if there are no complications, the case usually proceeds to satisfactory resolution. Meningitis is a fatal complication. Endocarditis is extremely grave, much more so than pericarditis, from which many cases recover. Early signs of heart-failure, dilatation of the right chamber, gradual cyanosis, and œdema of the lungs, are symptoms of the most serious character. As before stated, the danger of heart-clot is not great in pneumonia. The risk is in the extreme distention of the right chamber. I believe the firm fibrinous coagula entangled in the columnæ carneæ and the valves are invariably produced during the death agony. When there are symptoms of abscess of the lung or of gangrene the prognosis is extremely bad; yet cases are on record of recovery from both these conditions. Increasing rapidity of respiration, with difficulty in expectoration, very liquid and dark sputa, a low muttering delirium, dry tongue, and failing pulse, with a suffused lividity of the face, are indicative of approaching dissolution. Death rarely occurs from direct interference with the function of respiration, though it may happen in cases of extensive double pneumonia. In a majority of cases the fatal result is brought about by gradual heart-failure, whether induced by the prolonged action of the fever, the specific action of the poison, or paralysis due to overdistension of the right ventricle. A collateral œdema of the uninvolved portion of the lung, so much spoken of, rarely, I believe, occurs in pneumonia; nor is it likely, if the observations of Welch upon the production of this condition are correct, that in the course of pneumonia the left ventricle can be disproportionately weak in comparison with the right.

Termination.—*Resolution*, the process by which the lung is restored to its normal state, is effected partly by expectoration and partly by liquefaction and absorption of the exudate. It is not always possible to estimate the share respectively taken by these processes. There are cases in which a rapid resolution of extensive consolidation takes place without any special increase in the expectoration; and, on the other hand, during

resolution it is not uncommon to find in the expectoration the little plugs of fibrin and leucocytes which have been loosened from the air-cells and expelled by coughing. In a majority of cases both processes are probably at work. A variable time is taken in the restoration of the lung. Sometimes within a week or ten days the dulness is greatly diminished, the breath-sounds become clear, and, so far as physicial signs are any guide, the lung seems perfectly restored. It is to be remembered that in any case of pneumonia with extensive pleurisy a certain amount of dulness will persist for months, owing to thickening of the pleura. *Delayed resolution* is a condition which causes much anxiety to the physician. It may be postponed until the fourth, eighth, or even the tenth week. Usually the fever subsides, but the consolidation of the lung may persist, with great improvement in the general condition of the patient. In apex pneumonia the resolution is more apt to be retarded. It has been stated that bleeding is one cause of delayed resolution. A solid exudation can persist for weeks and yet the integrity of the lung may be ultimately restored. Grissole describes the lung from a patient who died on the sixtieth day in which the affected part looked not unlike the acute disease.

Abscess may result from purulent infiltration of the lung tissue. It occurred in 4 of my 100 cases. Usually the lung breaks in limited areas and the abscesses are not large, but they may involve a considerable portion of a lobe. This most serious complication is indicated by cavernous signs and the expectoration of purulent material containing elastic tissue. The constitutional symptoms are usually very severe. In a large majority of the clinical cases in which abscess of the lung is believed to follow an acute pneumonia, the process has in reality been rapid tuberculous consolidation with breaking of the lung tissue. There can, however, be no reasonable doubt that abscess of the lung does occur as a rare sequence of ordinary pneumonia.

Gangrene.—The presence of this complication is rendered evident by the horribly fetid odor of the expectoration, the presence of lung tissue, and crystals of fatty acids. It occurred in 3 of my 100 autopsies.

Fibroid Induration.—That a chronic interstitial pneumonia may follow the ordinary acute disease cannot be questioned, though it is probably the rarest of all terminations. It was present in one of my 100 autopsies. The patient, aged fifty-eight, died on the thirty-second day after the initial chill. The right lung was uniformly solid, grayish in color, firm, and presented in places a translucent, smooth, homogeneous aspect. In these areas the alveolar walls were thickened and the fibrinous plugs filling the air-cells were undergoing transformation into a new growth of connective tissue.

Mortality.—Pneumonia is one of the most fatal of acute diseases. Hospital statistics show that the mortality ranges from twenty to forty per cent Of 1,012 cases at the Montreal General Hospital, the mortality was 20·4 per cent. It appears to be somewhat more fatal in southern

climates. Of 3,969 cases treated at the Charity Hospital, New Orleans, the death rate was 28·01 per cent. It has been urged that the mortality in this disease has been steadily increasing, and attempts have been made to connect this increase with the expectant plan of treatment at present in vogue. But the careful and thorough analysis by C. N. Townsend and A. Coolidge, Jr.,* of 1,000 cases at the Massachusetts General Hospital indicates clearly that, when all circumstances are taken into consideration, this conclusion is not justified. They found that when all fatal cases over fifty years of age were omitted, and those patients who were delicate, intemperate, or the subject of some complication, there was very little variation from decade to decade, and that, excluding these cases, the rate was but little over ten per cent. In answer to the assertion that the modified treatment is in part responsible for the increased mortality, these authors show clearly that the rise in death rate took place in the period prior to 1860, when the treatment was entirely or in great part heroic.

According to the recent analysis of 708 cases at St. Thomas's Hospital by Hadden, H. W. G. McKenzie, and W. W. Ord, the mortality progressively increases from the twentieth year, rising from 3·7 per cent under that age to 22 per cent in the third decade, 30·8 per cent in the fourth, 47 per cent in the fifth, 51 per cent in the sixth, 65 per cent in the seventh decade.

Diagnosis.—No disease is more readily recognized in a large majority of the cases. The external characters, the sputa, and the physical signs combine to make one of the clearest of clinical pictures. After a study in the post-mortem room of my own and others' mistakes, I think that the ordinary lobar pneumonia of adults is rarely overlooked. Judging from my autopsy records, I should say that errors are particularly liable to occur in the intercurrent pneumonias, in those complicating chronic affections, and in the disease as met with in children, the aged, and drunkards. Tuberculo-pneumonic phthisis is frequently confounded with pneumonia. Pleurisy with effusion is, I believe, not often mistaken except in children.

In diabetes, Bright's disease, chronic heart-disease, pulmonary phthisis, and cancer, an acute pneumonia often ends the scene, and is frequently overlooked. In these cases the temperature is perhaps the best index, and should, more particularly if cough develops, lead to a careful examination of the lungs. The absence, however, of expectoration and sometimes the entire absence of pulmonary symptoms makes a diagnosis very difficult.

In children there are two special sources of error; the disease may be entirely masked by the cerebral symptoms and the case mistaken for one of meningitis. It is remarkable in these cases how few indications there are of pulmonary trouble. The other condition is pleurisy with effusion,

* Boston Medical and Surgical Journal, 1889.

which in children often has deceptive physical signs. The breathing may be intensely tubular and tactile fremitus may be present. The exploratory needle is sometimes required to decide the question. In the old and debilitated a knowledge that the onset of pneumonia is insidious, and that the symptoms are ill-defined and latent, should place the practitioner on his guard and make him very careful in the examination of the lungs in doubtful cases. In chronic alcoholism the cerebral symptoms may predominate and completely mask the local disease. As mentioned, the disease may assume the form of violent mania, but more commonly the symptoms are those of delirium tremens. In any case rapid pulse, rapid respiration, and fever are symptoms which should invariably excite suspicion of inflammation of the lungs.

Pneumonia is rarely confounded with ordinary consumption, but to differentiate acute tuberculo-pneumonic phthisis is often difficult. The case may set in with a chill. It may be impossible to determine which condition is present until softening occurs and elastic tissue and tubercle bacilli appear in the sputum. A similar mistake is sometimes made in children. With typhoid fever, pneumonia is not infrequently confounded. There are instances of pneumonia with the local signs well marked in which the patient rapidly sinks into what is known as the typhoid state, with dry tongue, rapid pulse, and diarrhœa. Unless the case is seen from the outset it may be very difficult to determine the true nature of the malady. On the other hand, there are cases of typhoid fever which set in with symptoms of lobar pneumonia—the so-called pneumo-typhus. It may be impossible to make a differential diagnosis in such a case unless the characteristic eruption develops.

Treatment.—Pneumonia is a self-limited disease, and runs its course uninfluenced in any way by medicine. It can neither be aborted nor cut short by any known means at our command. Even under the most unfavorable circumstances it will terminate abruptly and naturally, without a dose of medicine having been administered. A patient was admitted into one of my wards at the Philadelphia Hospital on the evening of the seventh day after the chill, in which he had been seen by one of my assistants, who had ordered him to go to hospital. He remained, however, in his house alone, without assistance, taking nothing but a little milk and bread and whisky, and was brought into the hospital by the police in a condition of active delirium. That night his temperature was 105° and his pulse above 120. In his delirium he came near escaping through the window of the ward. The following morning—the eighth day—the crisis occurred, and at ward class his temperature was below 98°. The entire lower lobe of the right side was found involved, and he entered upon a rapid convalescence. So also, under the favoring circumstances of good nursing and careful diet, the experience of many physicians in different lands has shown that pneumonia runs its course in a definite time, aborting sometimes spontaneously on the third or the fifth day, or continuing until the tenth or twelfth.

We have, then, no specific treatment for pneumonia. In cases of moderate severity a purely expectant plan may be followed—keeping the bowels open, regulating the diet, and, if necessary, giving a Dover's powder at night to procure sleep. In severer cases a symptomatic plan of treatment should be pursued, meeting the indications as they arise. The first distressing symptom is usually the pain in the side, which may be relieved by local depletion—by cupping or leeching—or, better still, by a hypodermic injection of morphia. In many cases the question comes up at the outset as to the propriety of venesection. The reproach of Van Helmont, that "a bloody Moloch presides in the chairs of medicine," cannot be brought against the present generation of physicians. During the first five decades of this century the profession bled too much, but during the last decades we have certainly bled too little. Pneumonia is one of the diseases in which a timely venesection may save life. To be of service it should be done early. In a full-blooded, healthy man with high fever and bounding pulse the abstraction of from twenty to thirty ounces of blood is in every way beneficial, relieving the pain and dyspnœa, reducing the temperature, and allaying the cerebral symptoms, so violent in some instances. Unfortunately, in a majority of the cases, bleeding is now used at a late stage in the disease, when the heart is beginning to fail, the right chambers are dilated, the face is of a dusky hue, the respirations are very rapid, and there are signs, perhaps, of œdema of the uninvolved portions of the lungs. Though resorted to rather as a forlorn hope, it is a rational practice, and, in cases of emphysema and of heart-disease, proves satisfactory under identical hydraulic indications, but, unfortunately, in a majority of the cases of pneumonia it proves futile. Time and again, in such cases, have I urged free venesection, but in twelve hospital patients bled under these circumstances only one recovered.

In the majority of cases requiring treatment the indications are to lower the temperature and to support the heart.

Fever alone is not, I think, hurtful; but it is difficult to differentiate the effects of fever and of the poisons circulating in the blood. It is not impossible, as some suppose, that the fever may be directly beneficial; still, high and prolonged pyrexia is undoubtedly dangerous and should be combated. Of efficient measures cold unquestionably heads the list. Perhaps the most convenient way is the application of ice-bags to the affected side—a practice long followed in Germany and now becoming prevalent in England and America. When the temperature is above 103° or 103·5° sponging may be resorted to. If the high fever is combined with brain symptoms the bath at 70° may be used without risk.

The use of medicinal antipyretics in pneumonia is of doubtful propriety. Quinine has been much vaunted. Personally I cannot speak of any special advantages which I have seen from its use. From thirty to sixty grains daily will reduce the temperature, in a certain proportion of the cases, one or two degrees, but in this respect it is far below other antipy-

retics. It is also not without ill effects in disturbing digestion or even causing vomiting, and, according to some writers—though this I have never noticed—inducing marked cardiac weakness and depression. Antipyrin, antifebrin, and phenacetin have had a thorough trial in pneumonia, and, although they still have their advocates, the general opinion of clinical physicians seems decidedly against their systematic employment.

The progressive cardiac weakness is, after all, the most important enemy to fight in pneumonia and is emphasized by the old axiom, *Sine pulsu nulla therapeia.* Doubtless this is in part caused by the fever, but much more important is the toxic action of the poisons produced in the course of the disease. To these must be added the third factor, overdistention of the right chambers of the heart. We are still without an agent which can counteract the gradual influence of the poisons which develop in the course of acute febrile diseases, such as typhoid fever, pneumonia, and diphtheria, the chief effect of which is exercised upon the circulation, increasing the rapidity of the pulse and inducing a progressive heart-failure. To meet this indication the general experience of physicians still points to alcohol as the most trustworthy remedy. Although some hold that alcohol in this condition is not indicated, I believe that it is in many instances the only remedy capable of tiding the patient over the most dangerous period. It should be given when the pulse becomes small, frequent, and feeble, or very compressible, and when the heart-sounds—particularly the second pulmonic sound—begin to lose their force. The amount will vary with the age of the patient and with his habits. Beginning with four to six ounces in the day the quantity may be increased, if necessary, to twelve or sixteen or even twenty ounces.

Of medicinal agents strychnine is one of the most valuable and has come into favor as a useful cardiac tonic. It may be given in doses of from a thirtieth to a twentieth of a grain. No certainty has as yet been reached as to the value of digitalis in the failing heart of fever. The practice is very general, but it is a drug to be used with caution in this condition. When there are signs of sudden or rapid heart-failure, hypodermic injections of ether will sometimes prove most serviceable. Of other stimulants ammonia is one of the most valuable and is best given in the form of the aromatic spirits, which is quite as satisfactory and much less nauseous than the usually administered carbonate of ammonia. Camphor and musk may also be employed.

Following the practice which is employed in spreading erysipelas, some writers have recommended direct antiseptic injections into the lung tissue itself. Lepine has used with benefit very dilute bichloride injections. In cases of gangrene following pneumonia, it might be of advantage to use iodoform oil or bichloride solutions.

The question of the use of arterial sedatives has not yet been settled. Aconite and veratrum viride and tartar emetic are largely used and loudly recommended by many physicians. I have never seen such benefit from

their early use as would warrant a recommendation, and when an arterial sedative is indicated in the robust, full-blooded, healthy individual, I much prefer the lancet.

Expectorants are rarely of any value in pneumonia. If any one wishes to be convinced of the futility of such remedies, let him study their action on a series of cases of sthenic pneumonia, in which it would be a real gain to loosen the cough and give to the sputa a certain degree of fluidity. Nor in the stage of resolution can they be said to be of any special service. In cases of tardy resolution I have not hesitated to use pilocarpine, as suggested by Riess.

For the distressing cough and the pain in the side, opium in some form may be given, either the hypodermic of morphia or, for the cough alone, Dover's powder. There has been a feeling in the profession that opium was counter-indicated in pneumonia, but I fully agree with Loomis that it may be given with safety and with the greatest comfort to the patient. With marked cerebral symptoms an ice-cap may be used. If there is delirium, the patient should be carefully watched. For these symptoms the cold bath is by far the most efficient remedy, and it or the cold pack should be resorted to without hesitation. For the complications, in the more serious ones, such as meningitis and endocarditis, but little can be done. Pleurisy with large effusion may require aspiration. If there is doubt as to the existence of fluid the exploratory needle should be used. It may be necessary, in pericarditis with extensive effusion, to aspirate the sac.

Careful feeding forms an essential part of the treatment. The diet should be light and made up of articles which, while nourishing, are not heavy and not apt to induce flatulency. Milk or milk-whey, broths, beef-juice, and eggs constitute the main articles of food. The starchy articles, as a rule, should be excluded, because they tend to induce flatulency. If the milk also has this effect, it is better to use the whey and egg-white or beef-juices. Before leaving the question of diet it may be mentioned that the use of cold drinks, such as soda or Apollinaris water, not only gives relief to the distressing thirst, but also helps to reduce the fever, and may diminish slightly the viscidity of the expectoration.

III. CHRONIC INTERSTITIAL PNEUMONIA

(Cirrhosis of Lung).

This consists in the gradual substitution to a greater or less extent of connective tissue for the normal lung. It is a fibroid change which may have its starting point in the tissue about the bronchi and blood-vessels, the interlobular septa, the alveolar walls, or in the pleura. So diverse are the different forms and so varied the conditions under which this change occurs that a proper classification is extremely difficult. We may recognize, however, two chief forms—the *local*, which involves only a limited

area of the lung substance, and the *diffuse*, invading either both lungs or an entire organ.

Etiology.—*Local* fibroid change in the lungs is common. It is a constant accompaniment of tubercle and in every case of phthisis the chronic interstitial changes play a very important *rôle*. In tumors, abscess, gummata, hydatids, and emphysema it also occurs. Fibroid processes are frequently met with at the apices of the lung and may be due either to a limited healed tuberculosis, to fibroid induration in consequence of pigment, or, in a few instances, may result from thickening of the pleura.

Diffuse Interstitial Pneumonia is met with under the following circumstances: 1. As a sequence of acute fibrinous pneumonia. Although extremely rare, this is recognized as a possible termination. From unknown causes resolution fails to take place. A gradual process of organization goes on in the fibrinous plugs within the air-cells and the alveolar walls become greatly thickened by a new growth, first of nuclear and subsequently fibrillated connective tissue. Macroscopically there is produced a smooth, grayish, homogeneous tissue which has the peculiar translucency of all new-formed connective tissue. This has been called gray induration. The subsequent history of this form needs more careful study. A majority of the cases terminate within a few months, and instances which have been followed from the outset are very rare.

In one of Charcot's cases, quoted by Bastian, death occurred about three months and a half after the onset of the acute disease and the lung was two thirds the normal size, grayish in color, and hard as cartilage. In the only case of the kind which has come under my observation, the patient died about a month from the onset of the chill. The lung was uniformly solid and grayish in color. In certain regions the fibrinous moulds in the air-cells were fatty, while in others there were areas of a grayish translucent aspect, firm, smooth, not at all granular, and resembling recent connective tissue. Microscopically, these areas showed advanced fibroid change and great thickening of the alveolar walls, while the fibrin plugs of the air-cells were undergoing fibroid transformation.

2. *Chronic Broncho-Pneumonia.*—The relation of broncho-pneumonia to cirrhosis of the lung has been specially studied by Charcot, who states that it may follow the acute or subacute form of this disease. The fibrosis extends from the bronchi, which are usually found dilated. The alveolar walls are thickened and the lobules converted into firm grayish masses, in which there is no trace of normal lung tissue. This process may go on and involve an entire lobe or even the whole lung. Many of these cases are tuberculous from the outset.

3. *Pleurogenous Interstitial Pneumonia.*—Charcot applies this term to that form of cirrhosis of the lung which follows invasion from the pleura. Doubt has been expressed by some writers whether this really occurs. While Wilson Fox is probably correct in questioning whether an

entire lung can become cirrhosed by the gradual invasion from the pleura, I think there can be no doubt that there are instances of primitive dry pleurisy, which, as Sir Andrew Clark has pointed out, gradually compresses the lung and at the same time leads to interstitial cirrhosis. This may be due in part to the fibroid change which follows prolonged compression. In some cases there seems to be a distinct connection between the greatly thickened pleura and the dense strands of fibrous tissue passing from it into the lung substance. Instances occur in which one lobe or the greater part of it presents, on section, a mottled appearance, owing to the increased thickness of the interlobular septa—a condition which may exist without a trace of involvment of the pleura. In many other cases, however, the extension seems to be so definitely associated with pleurisy that there is no doubt as to the causal connection between the two processes. In these instances the lung is removed with great difficulty, owing to the thickness and close adhesion of the pleura to the chest wall.

4. *Chronic Interstitial Pneumonia*, due to inhalation of dust. Zenker has proposed the term *pneumonokoniosis* for the group of diseases due to the irritating effects of dust in certain occupations, such as coal-mining, stone-cutting, axe-grinding, and working in iron dust. It is essentially a chronic broncho-pneumonia leading to fibroid induration, at first nodular and peribronchial, and finally involving large areas of the lung tissue, which are converted into dense grayish-brown or black masses. The subject will receive separate consideration.

The term cirrhosis should be limited strictly to those cases in which a lung is involved in the fibroid process, whether originating in the parenchyma or in the pleura. It should not be applied to fibroid phthisis of tuberculous origin.

Morbid Anatomy.—The disease is unilateral; the chest of the affected side is sunken, deformed, and the shoulder much depressed. On opening the thorax the heart is seen drawn far over to the affected side. The unaffected lung is emphysematous and covers the greater portion of the mediastinum. It is scarcely credible in how small a space, close to the spine, the cirrhosed lung may lie. Indeed, it may be overlooked, as happened in the case of a physician of my acquaintance, who left instructions that his lung should be sent to Palmer Howard, of Montreal. It was reported, however, that at the autopsy no lung could be found! The adhesions between the pleural membranes may be extremely dense and thick, particularly in the pleurogenous cases; but when the disease has originated in the lung there may be little thickening of the pleura. The organ is airless, firm, and hard. It strongly resists cutting, and on section shows a grayish fibroid tissue of variable amount, through which pass the blood-vessels and bronchi. The latter may be either slightly or enormously dilated. There are instances in which the entire lung is converted into a series of bronchiectatic cavities and the cirrhosis is apparent only in certain areas or at the root. The tuberculous cases can usually be

differentiated by the presence of an apical cavity, not bronchiectatic, and often large; and the other lung almost invariably shows tuberculous lesions. There are cases in which it is difficult to determine satisfactorily the true nature. A question of some interest in connection with chronic interstitial pneumonia is, Do softening and cavity formation ever occur apart from caseation and tuberculosis? That is to say, are there cavities in a cirrhotic lung which may be due to a simple necrosis? Undoubtedly, though they are rare; I have seen them in at least two instances of anthracosis, and Charcot* refers to them as "*ulcères du poumon*," to distinguish them from the abscess cavity of acute pneumonia or a tuberculous cavity. The other lung is always greatly enlarged and emphysematous. The heart is hypertrophied, particularly the right ventricle, and there may be marked atheromatous changes in the pulmonary artery. An amyloid condition of the viscera is found in some cases.

Symptoms and Course.—It is essentially a chronic disease, extending over a period of many years, and when once the condition is established the health may be fairly good. In a well-marked case the patient complains only of his chronic cough, perhaps of slight shortness of breath. In other respects he is quite well, and is usually able to do light work. The cases are commonly regarded as phthisical, though there may be scarcely a symptom of that affection except the cough. There are instances, however, of fibroid phthisis which cannot be distinguished from cirrhosis of the lung except by the presence of tubercle bacilli in the expectoration. As the bronchi are usually dilated, the symptoms and physical signs may be those of bronchiectasis. The cough is paroxysmal and the expectoration is generally copious and of muco-purulent or seropurulent nature. It is sometimes fetid. Hæmorrhage is by no means infrequent, and occurred in more than one half of the cases analyzed by Bastian. Walking on the level and in the ordinary affairs of life the patient may show no shortness of breath, but in the ascent of stairs and on exertion there may be dyspnœa.

Physical Signs.—*Inspection.*—The affected side is immobile, retracted, and shrunken, and contrasts in a striking way with the voluminous sound side. The intercostal spaces are obliterated and the ribs may even overlap. The shoulder is drawn down and from behind it is seen that the spine is bowed. The heart is greatly displaced, being drawn over by the shrinkage of the lung to the affected side. When the left lung is affected there may be a large area of visible impulse in the second, third, and fourth interspaces. Mensuration shows a great diminution in the affected side, and with the saddle-tape the expansion may be seen to be negative. The *percussion* note varies with the condition of the bronchi. It may be absolutely dull, particularly at the base or at the apex. In the axilla there may be a flat tympany or even an amphoric note over a large

* Œuvres complètes de J. M. Charcot, tome v, p. 139.

sacculated bronchus. On the opposite side the percussion note is usually hyperresonant. On *auscultation* the breath-sounds have either a cavernous or amphoric quality at the apex, and at the base are feeble, with mucous, bubbling râles. The voice-sounds are usually exaggerated. Cardiac murmurs are not uncommon, particularly late in the disease, when the right heart fails. These are, of course, the physical signs of the disease when it is well established. They naturally vary considerably, according to the stage of the process. The disease is essentially chronic, and may persist for fifteen or twenty years. Death occurs sometimes from hæmorrhage, more commonly from gradual failure of the right heart with dropsy, and occasionally from amyloid degeneration of the organs.

The *diagnosis* is never difficult. It may be impossible to say, without a clear history, whether the origin is pleuritic or pneumonic. Between cases of this kind and fibroid phthisis it is not always easy to discriminate, as the conditions may be almost identical. When tuberculosis is present, however, even in long-standing cases, bacilli are usually present in the sputa, and there may be signs of disease in the other lung.

Treatment.—It is only for an intercurrent affection or for an aggravation of the cough that the patient seeks relief. Nothing can be done for the condition itself. When possible the patient should live in a mild climate, and should avoid exposure to cold and damp. A distressing feature in some cases is the putrefaction of the contents of the dilated tubes, for which the same measures may be used as in fetid bronchitis.

IV. BRONCHO-PNEUMONIA (*Capillary Bronchitis*).

This is essentially an inflammation of the terminal bronchus and the air-vesicles which make up a pulmonary lobule, whence the term broncho-pneumonia. It is also known as lobular, in contradistinction to lobar pneumonia. The term catarrhal is less applicable. The process begins in all cases with an inflammation of the capillary bronchi, which is a condition rarely if ever found without involvement of the lobular structures, so that it is now customary to consider the affections together.

Etiology.—Broncho-pneumonia is as a rule a secondary affection met with under the following circumstances:

1. As a sequence of the infectious fevers—measles, diphtheria, whooping-cough, scarlet fever, and, less frequently, small-pox, erysipelas, and typhoid fever. In children it forms the most serious complication of these diseases, and in reality causes more deaths than are due directly to the fevers.* In large cities it ranks next in fatality to infantile diarrhœa. Following, as it does, the contagious diseases which principally affect children, we find that a large majority of cases occur during early l[illegible]

* Cyclopædia of the Diseases of Children, vol. ii.

According to Morrill's Boston statistics, it is most fatal during the first two years of life. The number of cases in a community increases or decreases with the prevalence of measles, scarlet fever, and diphtheria. It is most prevalent in the winter and spring months. In the febrile affections of adults broncho-pneumonia is not very common. Thus in typhoid fever it is not so frequent as lobar pneumonia, though isolated areas of consolidation at the bases are by no means rare in protracted cases of this disease. In old people it is an extremely common affection, following debilitating causes of any sort, and supervening in the course of chronic Bright's disease and various acute and chronic maladies.

2. In the second division of this affection are embraced the cases of so-called aspiration or deglutition pneumonia. Whenever the sensitiveness of the larynx is benumbed, as in the coma of apoplexy or uræmia, minute particles of food or drink are allowed to pass the *rima*, and, reaching finally the smaller tubes, excite an intense inflammation similar to the vagus pneumonia which follows the section of the pneumogastrics in the dog. Cases are very common after operations about the mouth and nose, after tracheotomy, and in cancer of the larynx and œsophagus. The aspirated particles in some instances induce such an intense broncho-pneumonia that suppuration or even gangrene supervenes.

3. The most common and fatal form of broncho-pneumonia is that excited by the tubercle bacillus, which has already been considered.

Among general predisposing causes may be mentioned age. As just noted, it is prone to attack infants, and a majority of cases of pneumonia in children under five years of age are of this form. At the opposite extreme of life it is also common, particularly in association with various debilitating circumstances and chronic diseases incident to the old. In children rickets and diarrhœa are marked predisposing causes, and broncho-pneumonia is one of the most frequent post-mortem-room lesions in infants' homes and foundling asylums. The disease prevails more extensively among the poorer classes, because their children are of necessity more exposed and cannot have the needful care and nursing, particularly after eruptive fevers.

Morbid Anatomy.—In the lungs of a child dead of broncho-pneumonia, after measles or diphtheria, the appearances are very characteristic. On the pleural surfaces, particularly toward the base, are seen depressed bluish or blue-brown areas of collapse, between which the lung tissue is of a lighter color. Here and there are projecting portions over which the pleura may be slightly turbid or granular. The lung is fuller and firmer than normal, and, though in great part crepitant, there can be felt in places throughout the substance solid, nodular bodies. The dark depressed areas may be isolated or a large section of one lobe may be in the condition of collapse or atelectasis. Gradual inflation by a blow-pipe inserted in the bronchus will distend a great majority of these collapsed areas. On section, the general surface has a dark reddish color and usu-

ally drips blood. Projecting above the level of the section are lighter red or reddish-gray areas representing the patches of broncho-pneumonia. These may be isolated and separated from each other by tracts of uninflamed tissue or they may be in groups or the greater part of a lobe may be involved. Study of a favorable section of an isolated patch shows: (*a*) A dilated central bronchiole full of tenacious purulent mucus. A fortunate section parallel to the long axis may show a racemose arrangement—the alveolar passages full of muco-pus. (*b*) Surrounding the bronchus for from 3 to 5 mm. or even more is an area of grayish-red consolidation, usually elevated above the surface and firm to the touch. Unlike the consolidation of lobar pneumonia, it may present a perfectly smooth surface, though in some instances it is distinctly granular. In a late stage of the disease small grayish-white points may be seen, which on pressure may be squeezed out as purulent droplets. A section in the axis of the lobule may present a somewhat grape-like arrangement, the stalk and stems representing the bronchioles and alveolar passages filled with a yellowish or grayish-white pus, while surrounding them is a reddish-brown hepatized tissue. (*c*) In the immediate neighborhood of this peribronchial inflammation the tissue is dark in color, smooth, airless, at a somewhat lower level than the hepatized portion, and differs distinctly in color and appearance from the other portions of the lung. This is the condition to which the term *splenization* has been given. It really represents a tissue in the early stage of inflammation, and it perhaps would be as well to give up the use of this term and also that of *carnification*, which is only a more advanced stage. The condition of collapse probably always precedes this, and it is difficult in some instances to tell the difference, as one shades into the other. In fact, collapse, splenization, and carnification may be said in broncho-pneumonia to be steps preliminary to the condition of actual hepatization.

While, in many cases, the areas of broncho-pneumonia present a reddish-brown color and are indistinctly granular, in others, particularly in adults, the nodules may resemble more closely gray hepatization and the air-cells are filled with a grayish, muco-purulent material. Minute hæmorrhages are sometimes seen in the neighborhood of the inflamed areas or on the pleural surfaces. Emphysema is commonly seen at the anterior borders and upper portions of the lung or in lobules adjacent to the inflamed ones. In many cases following diphtheria and measles the process is so extensive that the greater part of a lobe is involved, and it looks like a case of lobar hepatization. It has not, however, the uniformity of this affection and collapsed dark strands may be seen between extensive areas of hepatized tissue.

Practically, in the morbid anatomy of broncho-pneumonia in children we may recognize three groups of cases: (1) Those in which the bronchitis and bronchiolitis are most marked and in which there may be no definite consolidation and yet on microscopical examination many of the

alveolar passages and adjacent air-cells appear filled with inflammatory products. (2) The disseminated broncho-pneumonia, in which there are scattered areas of peribronchial hepatization with patches of collapse, while a considerable proportion of the lobe is still crepitant. This is by far the most common condition. (3) Pseudo-lobar form, in which the greater portion of the lobe is consolidated, but not uniformly, for intervening strands of dark congested lung tissue separate the groups of hepatized lobules.

In the secondary broncho-pneumonia of adults, it is generally the disseminated form which is seen.

Microscopically, a cross section of a small broncho-pneumonic focus shows the following changes: In the centre is a bronchus filled with a plug of exudation, consisting of leucocytes and swollen epithelium. Section in the long axis may show irregular dilatations of the tube. The bronchial wall is swollen and infiltrated with cells. Under a low power it is readily seen that the air-cells next the bronchus are most densely filled, while toward the periphery of the focus the alveolar exudation becomes less. The contents of the air-cells are made up of leucocytes and swollen endothelial cells in varying proportion. Red corpuscles are not often present and a fibrin network is rarely seen, though it may be present in some alveoli. In the swollen walls are seen distended capillaries and numerous leucocytes. As Delafield has pointed out, the interstitial inflammation of the bronchi and alveolar walls is a special feature of broncho-pneumonia which distinguishes it from the ordinary croupous form.

The histological changes in the aspiration or deglutition broncho-pneumonia differ from the ordinary post-febrile form in a more intense infiltration of the air-cells with leucocytes, producing suppuration and foci of softening, and even tending to gangrene.

Broncho-pneumonia may terminate (1) in *resolution*, which when it once begins goes on more rapidly than in fibrinous pneumonia. Broncho-pneumonia of the apices, in a child, persisting for three or more weeks, particularly if it follows measles or diphtheria, is often tuberculous. In these instances, when resolution is supposed to be delayed, caseation has in reality taken place. (2) In *suppuration*, which is rarely seen apart from the aspiration and deglutition forms, in which it is extremely common. (3) In *gangrene*, which occurs under the same conditions. (4) In *fibroid changes—chronic broncho-pneumonia*—a rare termination in the simple, a common sequence of the tuberculous disease. Formerly it was thought that one of the most common changes in broncho-pneumonia, particularly in children, was caseation; but this is really a tuberculous process, the natural termination of an originally specific broncho-pneumonia. It is of course quite possible that a broncho-pneumonia, simple in its origin, may subsequently be the seat of infection by the *bacillus tuberculosis*.

Symptoms.—Much confusion has arisen from the description of capillary bronchitis as a separate affection, whereas it is only a part, though a primary and important one, of broncho-pneumonia. At the outset it may be said that if in convalescence from measles or in whooping-cough a child has an accession of fever with cough, rapid pulse, and rapid breathing, and if, on auscultation, fine râles are heard at the bases, or widely spread throughout the lungs, even though neither consolidation nor blowing breathing can be detected, the diagnosis of broncho-pneumonia may safely be made. I have never seen in a fatal case after diphtheria or measles a capillary bronchitis as the sole lesion. The onset is rarely sudden, or with a distinct chill; but after a day or so of indisposition the child gets feverish and begins to cough and to get short of breath. The fever is extremely variable; a range of from 102° to 104° is common. The skin is very dry and pungent. The cough is hard, distressing, and may be painful. Dyspnœa gradually becomes a prominent feature. Expiration may be jerky and grunting. The respirations may rise as high as 60 or even 80 in the minute. Within the first forty-eight hours the percussion resonance is not impaired; the note, indeed, may be very full at the anterior borders of the lungs. On auscultation, many râles are heard, chiefly the fine subcrepitant variety, with sibilant rhonchi. There may really be no signs indicating that the parenchyma of the lung is involved, and yet even at this early stage, within forty-eight hours of the onset of the pulmonary symptoms, I have repeatedly, after diphtheria, found scattered nodules of lobular hepatization. Northrup,* in his thorough article on the subject, notes a case in which death occurred within the first twenty-four hours, and, in addition to the extensive involvement of the smaller bronchi, the intralobular tissue also was involved in places. The dyspnœa is constant and progressive and soon signs of deficient aëration of the blood are noted. The face becomes a little suffused and the finger-tips bluish. The child has an anxious expression and gradually enters upon the most distressing stage of asphyxia. At first the urgency of the symptoms is marked, but soon the benumbing influence of the carbon dioxide on the nerve-centres is seen and the child no longer makes strenuous efforts to breathe. The cough subsides and, with a gradual increase in lividity and a drowsy restlessness, the right ventricle becomes more and more distended, the bronchial râles become more liquid as the tubes fill with mucus, and death occurs from heart paralysis. These are the symptoms of a severe case of broncho-pneumonia, or what the older writers called *suffocative catarrh*.

The *physical signs* may at first be those of capillary bronchitis, as indicated by the absence of dulness, the presence of fine subcrepitant and whistling râles. In many cases death takes place before any definite pneumonic signs are detected. When these exist they are much more frequent

* Reference Handbook of the Medical Sciences, art. Broncho-Pneumonia.

at the bases, where there may be areas of impaired resonance or even of positive dulness. When numerous foci involve the greater part of a lobe the breathing may become tubular, but in the scattered patches of ordinary broncho-pneumonia, following the fevers, the breathing is more commonly harsh than blowing. In grave cases there is retraction of the base of the sternum and of the lower costal cartilages during inspiration, pointing to deficient lung expansion.

Diagnosis.—With lobar pneumonia it may readily be confounded if the areas of consolidation are large and merged together. It is to be remembered that broncho-pneumonia occurs chiefly in children under five years of age, whereas lobar pneumonia in children is much more common between the ages of five and fifteen. No writer has so clearly brought out the difference between pneumonia at these periods as Gerhard,* of Philadelphia, whose papers on this subject, though published nearly sixty years ago, have the freshness and accuracy which characterize all the writings of that eminent physician. Holt has recently brought forward figures to show that lobar pneumonia is not infrequent in infants under two years of age. The mode of onset is essentially different in the two affections, the one developing insidiously in the course or at the conclusion of another disease, the other setting in abruptly in a child in good health. In lobar pneumonia the disease is almost always unilateral, in broncho-pneumonia bilateral. The chief trouble arises in cases of broncho-pneumonia, which by aggregation of the foci involves the greater part of one lobe. Here the difficulty is very great, and the physical signs may be practically identical, but in a broncho-pneumonia it is much more likely that a lesion will be found on the other side. The course of the two affections is very unlike; the lobar pneumonia in children terminates on the eighth or tenth day with abruptness, as in adults.

A still more difficult question to decide is whether an existing broncho-pneumonia is simple or tuberculous. In many instances the decision cannot be made, as the circumstances under which the disease occurs, the mode of onset, and the physical signs may be identical. It has often been my experience that a case has been sent down from the children's ward to the dead-house with the diagnosis of post-febrile broncho-pneumonia in which there was no suspicion of the existence of tuberculosis; but on section there were found tuberculous bronchial glands and scattered areas of broncho-pneumonia, some of which were distinctly caseous, while others showed signs of softening. I have already spoken fully of this in the section on tuberculosis, but it is well to emphasize the fact that there are many cases of broncho-pneumonia in children which time alone enables us to distinguish from tuberculosis. The existence of extensive disease at the apices or central regions is a suggestive indication, and signs of softening may be detected. In the vomited matter, which is brought

* American Journal of the Medical Sciences, vols. xiv and xv.

up after severe spells of coughing, sputum may be picked out and elastic tissue and bacilli detected.

It is a superfluous refinement to make a diagnosis between capillary bronchitis and catarrhal pneumonia, for the two conditions are part and parcel of the same disease. In simple bronchitis involving the larger tubes urgent dyspnœa and pulmonary distress are rarely present and the râles are coarser and more sibilant. It must not be forgotten that, as in lobar pneumonia, cerebral symptoms may mask the true nature of the disease, and may even lead to the diagnosis of meningitis. I recall more than one instance in which it could not be satisfactorily determined whether the infant had tuberculous meningitis or a cerebral complication of an acute pulmonary affection.

Prognosis.—In children enfeebled by constitutional disease and prolonged fevers broncho-pneumonia is terribly fatal, but in cases coming on in connection with whooping-cough or after measles recovery may take place in the most desperate cases. It is in this disease that the truth of the old maxim is shown—"Never despair of a sick child." The death-rate in children under five has been variously estimated at from thirty to fifty per cent. After diphtheria and measles thin, wiry children seem to stand broncho-pneumonia much better than fat, flabby ones. In adults the aspiration or deglutition pneumonia is a very fatal disease.

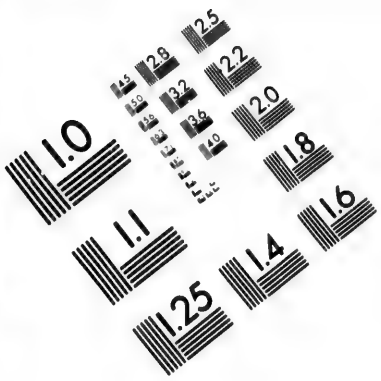
Prophylaxis.—Much can be done to reduce the probability of attack after febrile affections. Thus, in the convalescence from measles and whooping-cough, it is very important that the child should not be exposed to cold, particularly at night, when the temperature of the room naturally falls. In a nocturnal visit to the nursery—sometimes, too, I am sorry to say, to a children's hospital—how often one sees children almost naked, having kicked aside the bedclothes and having the night-clothes up about the arms! The use of light flannel "combinations" obviates this nocturnal chill, which is, I am sure, an important factor in the colds and pulmonary affections of young children, both in private houses and in institutions. The catarrhal troubles of the nose and throat should be carefully attended to, and during fevers the mouth should be washed two or three times a day with an antiseptic solution.

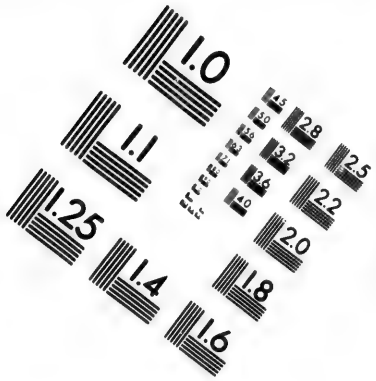
Treatment.—The frequency and the seriousness of broncho-pneumonia render it a disease which taxes to the utmost the resources of the practitioner. There is no acute pulmonary affection over which he at times so greatly despairs. On the other hand, there is not one in which he will be more gratified in saving cases which have seemed past all succor. The general arrangements should receive special attention. The room should be kept at an even temperature—about 65° to 68°—and the air should be kept moist with vapor.

At the outset the bowels should be opened by a mild purge, either castor oil or small doses of calomel, one twelfth to one sixth of a grain hourly until a movement is obtained, and care should be taken throughout

the attack to secure a daily movement. The common saline fever mixture of citrate of potash, liquor ammoniæ acetatis, and aromatic spirits of ammonia may be given every two or three hours. If the disease comes on abruptly with high fever, minim or minim and a half doses of the tincture of aconite may be given with it. The pain, the distressing symptoms, and the incessant cough often demand opium, which must of course be used with care and judgment in the case of young children, but which is certainly not contra-indicated and may be usefully given in the form of Dover's powder. Blisters are now rarely if ever employed, and even the jacket poultice has gone out of fashion. For the latter, however, I confess to a strong prejudice, and when lightly made and frequently changed it undoubtedly gives great relief. Much more commonly we now see, both in private and in hospital practice, the jacket of cotton-batting. Ice-poultices to the chest I have seen used apparently with great benefit, and they are warmly recommended by many German physicians as well as by Goodhart and others in England. The diet should consist of milk, broths, and egg albumen. Milk often curds and is disagreeable. Egg-white is particularly suitable and very acceptable when given in cold water with a little sugar. It forms, indeed, an excellent medium for the administration of the stimulants. If the pulse shows signs of failing, it is best to begin early with brandy. As in all febrile affections of children, cold water should be constantly at the bedside, and the child should be encouraged to drink freely. With these measures, in many cases the disease progresses to a favorable termination, but too often other and more serious symptoms arise. Cough becomes more distressing, dyspnœa increases, the ominous rattling of the mucus can be heard in the tubes, the child's color is not so good, and there is greater restlessness. Under these circumstances stimulant expectorants—ammonia, squills, and senega—should be given. Together they make a very disagreeable dose for a young child, particularly with the carbonate of ammonia. The aromatic spirits of ammonia is somewhat better. If the carbonate is employed, it must be given in small doses, not more than a grain to an infant of eighteen months. If the child has increasing difficulty in getting up the mucus, an emetic should be given—either the wine of ipecac or, if necessary, tartar emetic. There is no necessity, however, to keep the child constantly nauseated. Enough should be given to cause prompt emesis, and the benefit results in the expulsion of mucus from the larger tubes. In this stage, too, strychnine is undoubtedly helpful in stimulating the depressed respiratory centre. With commencing cyanosis, inhalations of oxygen may be employed, sometimes with great benefit.

With rapid failure of the heart, loud mucous rattles in the throat, and increasing lividity, every measure should be used to arouse the child and excite coughing. Alternate douches of hot and cold water, electricity, which I have seen applied with good results at Wiederhofer's clinic in Vienna, and hypodermic injections of ether may be tried. For the reduc-





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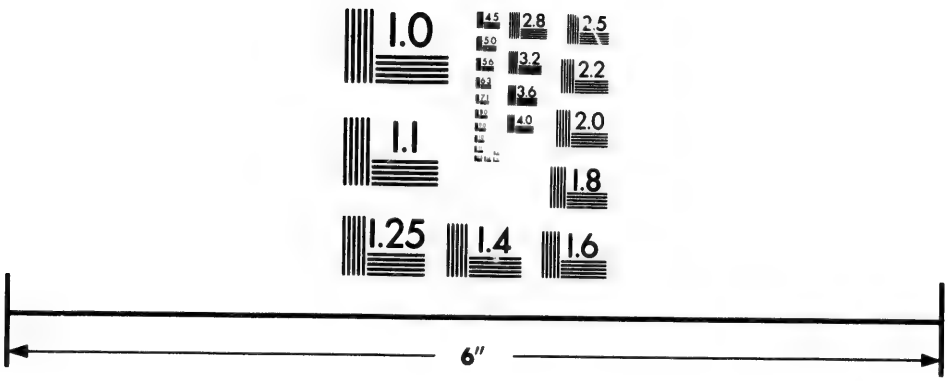

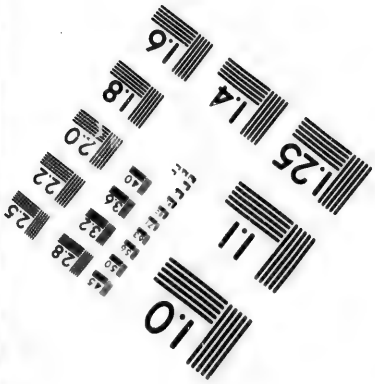

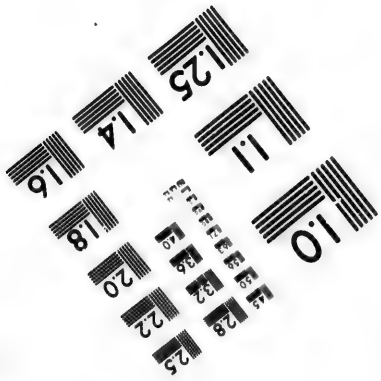

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tion of temperature, particularly if cerebral symptoms are prominent, there is nothing so satisfactory as the wet pack or the cold bath. In the case of children, when the latter is used it should be graduated, beginning with a temperature which is pleasantly warm and gradually reducing it to 75° or 80°. Even when the temperature is not high, the cerebral symptoms are greatly relieved by the bath or the pack.

V. EMPHYSEMA.

Rupture of superficial vesicles may produce pneumothorax. In the case of deep-seated alveoli the air escapes into the interlobular connective tissue and causes a condition comparable to ordinary subcutaneous emphysema. It is not a very serious condition and rarely produces symptoms. It usually results from violent expiratory efforts, as in whooping-cough. The air-bubbles escape into the interlobular tissue, in which they look like little rows of beads, and when extensive, the lobules are distinctly outlined by them (interstitial emphysema). There may be large bullæ beneath the pleura. A very rare event is the rupture close to the root of the lung and the passage of air along the trachea into the subcutaneous tissues of the neck.

The condition in which the infundibular passages and the alveoli are dilated is called *vesicular emphysema.*

A practical division may be made into compensatory, hypertrophic, and atrophic forms.

I. Compensatory Emphysema.

Whenever a region of the lung does not expand fully in inspiration, either another portion of the lung must expand or the chest wall sink in order to occupy the space. The former almost invariably occurs. We have already mentioned that in broncho-pneumonia there is a vicarious distention of the air-vesicles in the adjacent healthy lobules, and the same happens in the neighborhood of tuberculous areas and cicatrices. In general pleural adhesions there is often compensatory emphysema, particularly at the anterior margins of the lung. The most advanced example of this form is seen in cirrhosis, when the unaffected lung increases greatly in size, owing to distention of the air-vesicles. A similar though less marked condition is seen in extensive pleurisy with effusion and in pneumothorax.

At first, this distention of the air-vesicles is a simple physiological process and the alveolar walls are stretched but not atrophied. Ultimately, however, in many cases they waste and the contiguous air-cells fuse, producing true emphysema.

II. Hypertrophic Emphysema.

This form, also known as substantive or idiopathic emphysema, is a well-marked clinical affection, characterized by enlargement of the lungs, due to distention of the air-cells and atrophy of their walls, and clinically by imperfect aëration of the blood and more or less marked dyspnœa.

Etiology.—Emphysema is the result of persistently high intra-alveolar tension acting upon a congenitally weak lung tissue. If the mechanical views which have prevailed so long as to its origin were true, the disease would certainly be much more common; since violent respiratory efforts, believed to be the essential factor, are performed by a majority of the working classes. Strongly in favor of the view that the nutritive change in the air-cells is the primary factor is the markedly hereditary character of the disease and the frequency with which it starts early in life. These are two points upon which scarcely sufficient stress has been laid. To James Jackson, Jr., of Boston, we owe the first observations on the hereditary character of emphysema. Working under Louis's directions, he found that in 18 out of 28 cases one or both parents were affected.

I have been impressed by the frequency of the condition in children, and the number of cases in which on inquiry symptoms pointing to the occurrence of the disease in childhood can be obtained. It may develop, too, in several members of the same family. We are still ignorant as to the nature of this congenital pulmonary weakness. Cohnheim thinks it probably due to a defect in the development of the elastic-tissue fibres, a statement which is borne out by Eppinger's observations.

Heightened pressure within the air-cells may be due to forcible inspiration or expiration. Much discussion has taken place as to the part played by these two acts in the production of the disease. The inspiratory theory was advanced by Laennec and subsequently modified by Gairdner, who held that in the chronic bronchitis areas of collapse were induced, and compensatory distention took place in the adjacent lobules. This unquestionably does occur in the vicarious or compensatory emphysema, but it probably is not a factor of much moment in the form now under consideration. The expiratory theory, which was supported by Mendelsohn and Jenner, accounts for the condition in a much more satisfactory way. In all straining efforts and violent attacks of coughing, the glottis is closed and the chest walls are strongly compressed by muscular efforts, so that the strain is thrown upon those parts of the lung least protected, as the apices and the anterior margins, in which we always find the emphysema most advanced. The sternum and costal cartilages gradually yield to the heightened intrathoracic pressure and are, in advanced cases, pushed forward, giving the characteristic rotundity to the thorax. As mentioned, the cartilages gradually become calcified. One theory of

the disease is that there is a gradual enlargement of the thorax and the lungs increase in volume to fill up the space.

Of other etiological factors occupation is the most important. The disease is met with in players on wind instruments, in glass-blowers, and in occupations necessitating heavy lifting or straining. Whooping-cough and bronchitis play an important *rôle*, not so much in the changes which they induce in the bronchi as in consequence of the prolonged attacks of coughing.

Morbid Anatomy.—The thorax is capacious, usually barrel-shaped, and the cartilages are calcified. On removal of the sternum, the anterior mediastinum is found completely occupied by the edges of the lungs, and the pericardial sac may not be visible. The organs are very large and have lost their elasticity, so that they do not collapse either in the thorax or when placed on the table. The pleura is pale and there is often an absence of pigment, sometimes in patches, termed by Virchow *albinism* of the lung. To the touch they have a peculiar, downy, feathery feel, and pit readily on pressure. This is one of the most marked features. Beneath the pleura greatly enlarged air-vesicles may be readily seen. They vary in size from ½ to 3 mm., and irregular bullæ, the size of a walnut or larger, may project from the free margins. The best idea of the extreme rarefaction of the tissue is obtained from sections of a lung distended and dried. At the anterior margins the structure may form an irregular series of air-chambers, resembling the frog's lung. On careful inspection with the hand-lens, remnants of the interlobular septa or even of the alveoli may be seen on these large emphysematous vesicles. Though general throughout the organs, the distention is more marked, as a rule, at the anterior margins, and is often specially developed at the inner surface of the lobe near the root, where in extreme cases air-spaces as large as an egg may sometimes be found. Microscopically there is seen atrophy of the alveolar walls, by which is produced the coalescence of neighboring air-cells. In this process the capillary network disappears before the walls are completely atrophied. The loss of the elastic tissue is a special feature. It is stated, indeed, that in certain cases there is a congenital defect in the development of this tissue. The epithelium of the air-cells undergoes a fatty change, but the large distended air-spaces retain a pavement layer.

The bronchi in emphysema show important changes. In the larger tubes the mucous membrane may be rough and thickened from chronic bronchitis; often the longitudinal lines of submucous elastic tissue stand out prominently. In the advanced cases many of the smaller tubes are dilated, particularly when, in addition to emphysema, there are peribronchial fibroid changes. Bronchiectasis is not, however, an invariable accompaniment of emphysema, but, as Laennec remarks, it is difficult to understand why it is not more common. Of associated morbid changes the most important are found in the heart. The right chambers are

dilated and hypertrophied, the tricuspid orifice is large, and the valve segments are often thickened at the edges. In advanced cases the cardiac hypertrophy is general. The pulmonary artery and its branches may be wide and show marked atheromatous changes.

The changes in the other organs are those commonly associated with prolonged venous congestion.

Symptoms.—The disease may be tolerably advanced before any special symptoms develop. A child, for instance, may be somewhat short of breath on going up-stairs or may be unable to run and play as other children without great discomfort; or, perhaps, has attacks of slight lividity. Doubtless much depends upon the completeness of cardiac compensation. When this is perfect, there may be no special interruption of the pulmonary circulation and, except in violent exertion, there is no interference with the aëration of the blood. In well-developed cases the following are the most important symptoms: *Dyspnœa*, which may be felt only on slight exertion, or may be persistent, and aggravated by intercurrent attacks of bronchitis. The respirations are often harsh and wheezy, and expiration is distinctly prolonged.

Cyanosis of an extreme grade is more common in emphysema than in other affections with the exception of congenital heart-disease. So far as I know it is the only disease in which a patient may be able to go about and even to walk into the hospital or consulting-room with a lividity of startling intensity. The contrast between the extreme cyanosis and the comparative comfort of the patient is very striking. In other affections of the heart and lungs associated with a similar degree of cyanosis the patient is invariably in bed and usually in a state of orthopnœa.

Bronchitis with associated cough is a frequent symptom and often the direct cause of the pulmonary distress. The contrast between emphysematous patients in the winter and summer is marked in this respect. In the latter they may be comfortable and able to attend to their work, but with the cold and changeable weather they are laid up with attacks of bronchitis. Finally, in fact, the two conditions become inseparable and the patient has persistently more or less cough. The acute bronchitis may produce attacks not unlike asthma. In some instances this is true spasmodic asthma, with which emphysema is frequently associated.

As age advances and with successive attacks of bronchitis the condition gets slowly worse. In hospital practice it is common to admit patients over sixty with well-marked signs of advanced emphysema. The affection can generally be told at a glance—the rounded shoulders, barrel chest, the thin yet oftentimes muscular form, and sometimes, I think, a very characteristic facial expression.

There is another group, however, of younger patients from twenty-five to forty years of age who winter after winter have attacks of intense cyanosis in consequence of an aggravated bronchial catarrh. On inquiry we find that these patients have been short-breathed from infancy, and they

belong, I believe, to a category in which there has been a primary defect of structure in the lung tissue.

Physical Signs.—*Inspection.*—The thorax is markedly altered in shape; the antero-posterior diameter is increased and may be even greater than the lateral, so that the chest is barrel-shaped. The appearance is somewhat as if the chest was in a permanent inspiratory position. The sternum and costal cartilages are prominent. The lower zone of the thorax looks large and the intercostal spaces are much widened, particularly in the hypochondriac regions. The sternal fossa is deep, the clavicles stand out with great prominence, and the neck looks shortened from the elevation of the thorax and the sternum. A zone of dilated venules may be seen along the line of attachment of the diaphragm. Though this is common in emphysema, it is by no means peculiar to it. Andrew, of Bartholomew's Hospital, and, according to Duckworth, Laycock have called attention to it. This network in the lower thoracic region, just above the costal margin and following its curves, is a well-marked feature in many persons, and is seen not only in emphysema, but in many cases of hepatic trouble.

Behind, the curve of the spine is increased and the back is remarkably rounded, so that the scapulæ seem to be almost horizontal. Mensuration shows the rounded form of the chest; the antero-posterior diameter may exceed the transverse. The respiratory movements, which may look energetic and forcible, exercise little or no influence. The chest does not expand, but there is a general elevation. The inspiratory effort is short and quick; the expiratory movement is prolonged. There may be retraction instead of distention in the upper abdominal region during inspiration, and there is sometimes seen a transverse curve crossing the abdomen at the level of the twelfth rib. The apex beat of the heart is not visible, and there is usually marked pulsation in the epigastric region. The cervical veins stand out prominently and may pulsate.

Palpation.—The vocal fremitus is somewhat enfeebled but not lost. The apex beat can rarely be felt. There is a marked shock in the lower sternal region and very distinct pulsation in the epigastrium. *Percussion* gives greatly increased resonance, full and drum-like—what is sometimes called hyperresonance. The note is not often distinctly tympanitic in quality. The percussion note is greatly extended, the heart dulness may be obliterated, the upper limit of liver dulness is greatly lowered, and the resonance may extend to the costal margin. Behind, a clear percussion note extends to a much lower level than normal. The level of splenic dulness, too, may be lowered.

On *auscultation* the breath-sounds are usually enfeebled and may be masked by bronchitic râles. The most characteristic feature is the prolongation of the expiration, and the normal ratio may be reversed—4 to 1 instead of 1 to 4. It is often wheezy and harsh and associated with coarse râles and sibilant rhonchi. It is said that in interstitial emphysema there

may be a friction sound heard not unlike that of pleurisy. As already noted, the cardiac impulse may be barely felt in the lower sternal region. The heart-sounds are usually clear; but in advanced cases, when there is marked cyanosis, a tricuspid regurgitant murmur may be heard. Accentuation of the pulmonary second sound is present.

The *course* of the disease is slow but progressive, the recurring attacks of bronchitis aggravating the condition. Death may occur from intercurrent pneumonia, either lobar or lobular, and dropsy may supervene from cardiac failure. Occasionally death results from overdistention of the heart, with extreme cyanosis. Duckworth has called attention to fatal hæmorrhage in emphysema. It certainly is not common. In an old emphysematous patient at the Montreal General Hospital death followed the erosion of a main branch of the pulmonary artery by an ulcer near the bifurcation of the trachea.

Treatment.—Practically, the measures mentioned in connection with bronchitis should be employed. No remedy is known which has any influence over the progress of the condition itself. Bronchitis is the great danger of these patients, and therefore when possible they should live in an equable climate. In consequence of the venous engorgement they are liable to gastric and intestinal disturbance, and it is particularly important to keep the bowels regulated and to avoid the flatulency which often seriously aggravates the dyspnœa. Patients who come into the hospital in a state of urgent dyspnœa and lividity, with great engorgement of the veins, particularly if they are young and vigorous, should be bled freely. On more than one occasion I have saved the lives of persons in this condition by venesection. Inhalation of oxygen may be used and the remedies given already mentioned in connection with bronchitis. Strychnine will be found specially useful.

III. Atrophic Emphysema.

This is really a senile change and is called by Sir William Jenner small-lunged emphysema. It is really a primary atrophy of the lung, coming on in advanced life, and scarcely constitutes a special affection. It occurs in "withered-looking old persons" who may perhaps have had a winter cough and shortness of breath for years. In striking contrast to the essential or hypertrophic emphysema, the chest in this form is small. The ribs are obliquely placed, the decrease in the diameter being due to greatly increased obliquity in the position of the ribs. The thoracic muscles are usually atrophied. In advanced cases of this affection the lung presents a remarkable appearance, being converted into a series of large vesicles, on the walls of which the remnants of air-cells may be seen. It is a condition for which nothing can be done.

VI. GANGRENE OF THE LUNG.

Etiology.—Gangrene of the lung is not an affection *per se*, but occurs in a variety of conditions when necrotic areas undergo putrefaction. It is not easy to say why sphacelus should occur in one case and not in another, as the germs of putrefaction are always in the air-passages, and yet necrotic territories rarely become gangrenous. Total obstruction of a pulmonary artery, as a rule, causes infarction, and the area shut off does not often, though it may, sphacelate. Another factor would seem to be necessary—probably a lowered tissue resistance, the result of general or local causes. It is met with (1) as a sequence of lobar pneumonia. This rarely occurs in a previously healthy person—more commonly in the debilitated or in the diabetic subject. (2) Gangrene is very prone to follow the aspiration pneumonia, since the foreign particles rapidly undergo putrefactive changes. Of a similar nature are the cases of gangrene due to perforation of cancer of the œsophagus into the lung or into a bronchus. (3) The putrid contents of a bronchiectatic, more commonly of a tuberculous, cavity may excite gangrene in the neighboring tissues. The pressure bronchiectasis following aneurism or tumor may lead to extensive sloughing. (4) Gangrene may follow simple embolism of the pulmonary artery. More commonly, however, the embolus is derived from a part which is mortified or comes from a focus of bone disease. Lastly, gangrene of the lung may occur in conditions of debility during convalescence from protracted fever—occasionally, indeed, without our being able to assign any reasonable cause.

Morbid Anatomy.—Laennec, who first accurately described pulmonary gangrene, recognized a diffuse and a circumscribed form. The former, though rare, is sometimes seen in connection with pneumonia, more rarely after obliteration of a large branch of the pulmonary artery. It may involve the greater part of a lobe, and the lung tissue is converted into a horribly offensive greenish-black mass, torn and ragged in the centre. In the circumscribed form there is well-marked limitation between the gangrenous area and the surrounding tissue. The focus may be single or there may be two or more. The lower lobe is more commonly affected than the upper, and the peripheral more than the central portion of the lung. A gangrenous area is at first uniformly greenish brown in color; but softening rapidly takes place with the formation of a cavity with shreddy, irregular walls and a greenish, offensive fluid. The lung tissue in the immediate neighborhood shows a zone of deep congestion, often consolidation, and outside this an intense œdema. In the embolic cases the plugged artery can sometimes be found. When rapidly extending, vessels may be opened and violent hæmorrhage ensue. Perforation of the pleura is not uncommon. The irritating decomposing material usually excites the most intense bronchitis. Embolic processes are not infrequent. There is a remarkable association in some cases between circumscribed

gangrene of the lung and abscess of the brain. I have seen two such cases. One of these, a young man, an Arab, was brought to the University Hospital, almost exsanguine from pulmonary hæmorrhage. He gradually recovered. There were very limited signs in the middle lobe of the right lung, which persisted, but no bacilli were found. There was no fetor of the breath. Weeks afterward he developed severe headache, and in a few days became comatose and died. There was a circumscribed area of healing gangrene at the margin of the l ng with great increase of fibrous tissue about it. The artery going to this somewhat wedge-shaped area was obliterated. The contents of the encapsulated cavity were very fetid. There was a large limited abscess in the parieto-temporal region on the right side.

Symptoms and Course.—Usually definite symptoms of local pulmonary disease precede the characteristic features of gangrene. These, of course, are very varied, depending on the nature of the trouble. The sputum is very characteristic. It is intensely fetid—usually profuse—and, if expectorated into a conical glass, separates into three layers—a greenish-brown, heavy sediment; an intervening thin liquid, which sometimes has a greenish or a brownish tint; and, on top, a thick, frothy layer. Spread on a glass plate, the shreddy fragments of lung tissue can readily be picked out. Microscopically, elastic fibres are found in abundance, with granular matter, pigment grains, fatty crystals, bacteria, and leptothrix. It is stated that elastic tissue is sometimes absent, but I have never met with such an instance. The peculiar plugs of sputum which occur in bronchiectasy are not found. Blood is often present, and, as a rule, is much altered. The sputum has, in a majority of the cases, an intensely fetid odor, which is communicated to the breath and may permeate the entire room. It is much more offensive than in fetid bronchitis or in abscess of the lung. The fetor is particularly marked when there is free communication between the gangrenous cavities and the bronchi. On several occasions I have found, post mortem, localized gangrene, which had been unsuspected during life, and in which there had been no fetor of the breath.

The physical signs, when extensive destruction has occurred, are those of cavity, but the limited circumscribed areas may be difficult to detect. Bronchitis is always present.

Among the general symptoms may be mentioned fever, usually of moderate grade; the pulse is rapid, and very often the constitutional depression is severe. But the only special features indicative of gangrene are the sputa and the fetor of the breath. The patient generally sinks from exhaustion. Fatal hæmorrhage may ensue. I have already mentioned a case in which a hæmorrhage from a circumscribed gangrene nearly proved fatal, and I have seen one fatal instance after pneumonia.

Treatment.—The treatment of gangrene is very unsatisfactory. The indications, of course, are to disinfect the gangrenous area, but this is

often impossible. An antiseptic spray of carbolic acid may be employed. A good plan is for the patient to use over the mouth and nose an inhaler, which may be charged with a solution of carbolic acid or creosote. If the signs of cavity are distinct an attempt should be made to cleanse it by direct injections of an antiseptic solution. If the patient's condition is good and the gangrenous region can be localized, an attempt should be made to treat it surgically. Successful cases have been reported. The general condition of the patient is always such as to demand the greatest care in the matter of diet and nursing.

VII. ABSCESS OF THE LUNG.

Etiology.—Suppuration occurs in the lung under the following conditions: (1) As a sequence of inflammation, either lobar or lobular. Apart from the purulent infiltration this is unquestionably rare, and even in lobar pneumonia the abscesses are of small size and usually involve, as Addison remarked, several points at the same time. On the other hand, abscess formation is extremely frequent in the deglutition and aspiration forms of lobular pneumonia. After wounds of the neck or operations upon the throat, in suppurative disease of the nose or larynx, occasionally even of the ear (Volkmann), infective particles reach the bronchial tubes by aspiration and excite an intense inflammation which often ends in suppuration. Cancer of the œsophagus, perforating the root of the lung or into the bronchi, may produce extensive suppuration. The abscesses vary in si m a walnut to an orange, and have ragged and irregular walls, and ent, sometimes necrotic, contents.

(2) Embolic, so-called metastatic, abscesses, the result of infectious emboli, are extremely common in a large proportion of all cases of pyæmia. They may occur in enormous numbers and present very definite characters. As a rule they are superficial, beneath the pleura, and often wedge-shaped. At first firm, grayish red in color, and surrounded by a zone of intense hyperæmia, suppuration soon follows with the formation of a definite abscess. The pleura is usually covered with greenish lymph, and perforation sometimes takes place with the production of pneumothorax.

(3) Perforation of the lung from without, lodgment of foreign bodies, and, in the right lung, perforation from abscess of the liver or suppurating echinococcus cyst are occasional causes of pulmonary abscess.

(4) Suppurative processes play an important part in chronic pulmonary tuberculosis, many of the symptoms of which are due to them.

Symptoms.—Abscess following pneumonia is easily recognized by an aggravation of the general symptoms and by the physical signs of cavity and the characters of the expectoration. Embolic abscesses cannot often be recognized, and the local symptoms are generally masked in the

general pyæmic manifestations. The characters of the sputum are of great importance in determining the presence of abscess. The odor is offensive, yet it rarely has the horrible fetor of gangrene or of putrid bronchitis. In the pus fragments of lung tissue can be seen, and the elastic tissue may be very abundant. The presence of this with the physical signs rarely leaves any question as to the nature of the trouble. Embolic cases usually run a fatal course. Recovery occasionally occurs after pneumonia.

Medicinal treatment is of little avail in abscess of the lung. When well defined and superficial, an attempt should always be made to open and drain it. A number of successful cases have already been treated in this way.

VIII. PNEUMONOKONIOSIS.

Under this term, introduced by Zenker, is embraced those diseases of the lungs due to the inhalation of dusts in various occupations. They have received various names, according to the nature of the inhaled particles—*anthracosis*, or coal-miner's disease; *siderosis*, due to the inhalation of metallic dusts, particularly iron; *chalicosis*, due to the inhalation of mineral dusts, producing the so-called stone-cutter's phthisis, or the "grinder's rot" of the Sheffield workers.

The dust particles inhaled into the lungs are dealt with extensively by the ciliated epithelium and by the phagocytes, which exist normally in the respiratory organs. The ordinary mucous corpuscles take in a large number of the particles, which fall upon the trachea and main bronchi. The cilia sweep the mucus out to a point from which it can be expelled by coughing. It is doubtful if the particles ever reach the air-cells, but the swollen alveolar cells (in which they are in numbers) probably pick them up on the way. The mucous and the alveolar cells are the normal respiratory scavengers. In dwellers in the country, in which the air is pure, they are able to prevent the access of dust particles to the lung tissue, so that even in adults these organs present a rosy tint, very different from the dark, carbonized appearance of the lungs of dwellers in cities. When the impurities in the air are very abundant, a certain proportion of the dust particles escapes these cells and penetrates the mucosa, reaching the lymph spaces, where they are attacked at once by the cells of the connective-tissue stroma, which are capable of ingesting and retaining a large quantity In coal-miners, coal-heavers, and others whose occupations necessitate the constant breathing of a very dusty atmosphere even these forces are insufficient. Many of the particles enter the lymph stream and, as Arnold has shown in his beautiful researches, are carried (1) to the lymph nodules surrounding the bronchi and blood-vessels; (2) to the interlobular septa beneath the pleura, where they lodge in and between the tissue elements; and (3) along the larger lymph channels to the substernal, bronchial and tracheal glands, in which the stroma cells of

the follicular cords dispose of them permanently and prevent them from entering the general circulation. Occasionally in anthracosis the carbon grains do reach the general circulation, and the coal dust is found in the liver and spleen. As Weigert has shown, this occurs when the densely pigmented bronchial glands closely adhere to the pulmonary veins, through the walls of which the carbon particles pass to the general circulation. The lung tissue has a remarkable tolerance for these particles, probably because a large proportion of them is warehoused, so to speak, in protoplasmic cells. By constant exposure a limit is reached, and there is brought about a very definite pathological condition, an interstitial sclerosis. In coal-miners this may occur in patches, even before the lung tissue is uniformly infiltrated with the dust. In others it appears only after the entire organs have become so laden that they are dark in color, and an ink-like juice flows from the cut surface. The lungs of a miner may be black throughout and yet show no local lesions and be everywhere crepitant.

As already mentioned, the particles are deposited in large numbers in the follicular cords of the tracheal and bronchial glands and of the peri-bronchial and peri-arterial lymph nodules, and in these they finally excite proliferation of the connective-tissue elements. It is by no means uncommon to find in persons whose lungs are only moderately carbonized the bronchial glands sclerosed and hard. In anthracosis the fibroid changes usually begin in the peri-bronchial lymph tissue, and in the early stage of the process the sclerosis may be largely confined to these regions. A Nova Scotian miner, aged thirty-six, died under my care, at the Montreal General Hospital, of black small-pox, after an illness of a few days. In his lungs (externally coal-black) there were round and linear patches ranging in size from a pea to a hazel-nut, of an intensely black color, airless and firm, and surrounded by a crepitant tissue, slate-gray in color. In the centre of each of these areas was a small bronchus. Many of them were situated just beneath the pleura, and formed typical examples of limited fibroid broncho-pneumonia. In addition there is usually thickening of the alveolar walls, particularly in certain areas. By the gradual coalescence of these fibroid patches large portions of the lung may be converted into firm grayish-black, in the case of the coal-miner—steel-gray, in the case of the stone-worker—areas of cirrhosis. In the case of a Cornish miner, aged sixty-three, who died under my care, one of these fibroid areas measured 18 by 6 cm. and 4·5 cm. in depth.

A second important factor in these cases is chronic bronchitis, which is present in a large proportion and really causes the chief symptoms. A third is the occurrence of emphysema, which is almost invariably associated with long-standing cases of pneumonokoniosis. With the changes so far described, unless the cirrhotic area is unusually extensive, the case may present the features of chronic bronchitis with emphysema, but finally another element comes into play. In the fibroid areas softening occurs,

probably a process of necrosis similar to that by which softening is produced in fibro-myomata of the uterus. At first these are small and contain a dark liquid. Charcot calls them, as already mentioned, *ulcères du poumon*. They rarely attain a large size unless a communication is formed with the bronchus, in which case they may become converted into suppurating cavities. The question has been much discussed of late as to what part the tubercle bacillus plays in these cases of pneumonokoniosis with cavity formation. In some instances there is certainly a tuberculous process ingrafted, but that large excavations may occur, or in other instances bronchiectasis without the presence of bacillis, I have convinced myself by the examination of several characteristic specimens.

The *siderosis* induced by the oxide of iron causes an interstitial pneumonia similar to anthracosis. Workers in brass and in bronze are liable to a similar affection.

Chalicosis, due to the deposit of particles of silex and alumina, is found in the makers of mill-stones, particularly the French mill-stones, and also in knife and axe grinders and stone-cutters. Anatomically, this form is characterized by the production of nodules of various sizes, which are cut with the greatest difficulty and sometimes present a curious grayish, even glittering, crystalloid appearance.

Workers in flax and in cotton, and grain-shovellers are also subject to these chronic interstitial changes in the lungs. In all these occupations, as shown by Greenhow, to whose careful studies we owe so much of our knowledge of these diseases, the condition of the lung may ultimately be almost identical.

The *symptoms* do not come on until the patient has worked for a variable number of years in the dusty atmosphere. As a rule there are cough and failing health for a prolonged period of time before complete disability. The coincident emphysema is responsible in great part for the shortness of breath and wheezy condition of these patients. The expectoration is usually muco-purulent, often profuse; in a case of anthracosis, very dark in color—the so-called "black spit"; in a case of chalicosis there may be seen under the microscope the bright angular particles of silica.

Even when there are physical signs of cavity tubercle bacilli are not necessarily, and indeed in my experience they are not usually present. It is remarkable for how long a time a coal-miner may continue to bring up sputum laden with coal particles even when there are only signs of a chronic bronchitis. Many of the particles are contained in the cells of the alveolar epithelium. In these instances it appears that an attempt is made by the leucocytes to rid the lungs of some of the carbon grains.

The *diagnosis* of the condition is rarely difficult; the expectoration is usually characteristic. It must always be borne in mind that chronic

bronchitis and emphysema form essential parts of the process and that in late stages there may be tuberculous infection.

The *treatment* of the condition is practically that of chronic bronchitis and emphysema.

IX. NEW GROWTHS IN THE LUNGS.

Etiology and Morbid Anatomy.—While primary tumors are rare, secondary growths are not uncommon.

The primary growths of the lung are either encephaloid, scirrhus or epithelioma. Recent observations show that the latter is the most common form. Sarcoma also is occasionally found as a primary growth, and still more rarely enchondroma.

The secondary growths may be of various forms. Most commonly they follow tumors in the digestive or genito-urinary organs; not infrequently also tumors of the bone. There may be encephaloid, scirrhus, epithelioma, colloid, melano-sarcoma, enchondroma, or osteoma.

Primary cancer or sarcoma usually involves only one lung. The secondary growths are distributed in both. The primary growth generally forms a large mass, which may occupy the greater part of a lung. Occasionally the secondary growths are solitary and confined chiefly to the pleura, as in a remarkable example which came under my observation, in which the disease was secondary to a myelo-sarcoma of the wrist. The tumor mass occupied a large portion of the left side of the thorax. It grew from the pleura and extended only slightly into the lung, which was compressed and airless. The metastatic growths are nearly always disseminated. Occasionally they occupy a large portion of the pulmonary tissue. In a case of colloid cancer secondary to cancer of the pancreas, I found both lungs voluminous, heavy, only slightly crepitant, and occupied by circular translucent masses, varying in size from a pea to a large walnut.

There are numerous accessory lesions in the pulmonary new growths. There may be pleurisy, either cancerous or sero-fibrinous. The effusion may be hæmorrhagic, but in 200 cases of cancer, primary or secondary, of the lungs and pleura analyzed by Moutard-Martin, hæmorrhagic effusion occurred in only twelve per cent. The tracheal and bronchial glands are usually affected, the cervical glands not infrequently, and occasionally even the inguinal.

The disease is most common in the middle period of life. The primary form affects the sexes equally, but secondary cancer is much more frequent in women than in men. The conditions which predispose to it are quite unknown. It is a remarkable fact that the workers in the Schneeberg cobalt mines are very liable to primary cancer of the lungs. It is stated that in this region a considerable proportion of all deaths in persons over forty are due to this disease.

Symptoms.—The clinical features of neoplasms of the lungs are by no means distinctive, particularly in the case of primary growths. The patient may, indeed, as noted by Walshe, present no symptoms pointing to intrathoracic disease. Among the more important symptoms are pain, particularly when the pleura is involved; dyspnœa, which is apt to be paroxysmal when due to pressure upon the trachea; cough, which may be dry and painful and accompanied by the expectoration of a dark mucoid sputum. This so-called prune-juice expectoration, which was present ten times in eighteen cases of primary cancer of the lung, was thought by Stokes to be of great diagnostic value.

In many instances there are signs of compression of the large veins, producing lividity of the face and upper extremities, or occasionally of only one arm. Compression of the trachea and bronchi may give rise to urgent dyspnœa. The heart may be pushed over to the opposite side. The pneumogastric and recurrent laryngeal nerves are occasionally involved in the growth.

Physical Signs.—The patient, according to Walshe, usually lies on the affected side. On inspection this side may be enlarged and immobile and the intercostal spaces are obliterated. This is more commonly due to the effusion than to the growth itself. The external lymph-glands may be enlarged, particularly the clavicular. The signs, on percussion and auscultation, are varied, depending much upon the presence or absence of fluid. Signs of consolidation are, of course, present; the tactile fremitus is absent and the breath-sounds are usually diminished in intensity. Occasionally there is typical bronchial breathing. Among other symptoms may be mentioned fever, which is present in a certain number of cases. Emaciation is not necessarily extreme. The duration of the disease is from six to eight months. Occasionally the disease runs a very acute course, as noted by Carswell. Cases are reported in which death occurred in a month or six weeks, and in one instance—Jaccoud—the patient died in a week from the onset of the symptoms.

Diagnosis.—In secondary growths this is not difficult. The development of pulmonary symptoms within a year or two after the removal of a cancer of the breast, or after the amputation of a limb for osteo-sarcoma, or the onset of similar symptoms in connection with cancer of the liver, or of the uterus, or of the rectum, would be extremely suggestive. In primary cases the unilateral involvement, the anomalous character of the physical signs, the occurrence of prune-juice expectoration, the progressive wasting, and the secondary involvement of the cervical glands are the important points in the diagnosis.

V. DISEASES OF THE PLEURA.

I. ACUTE PLEURISY.

Anatomically, the cases may be divided into dry or adhesive pleurisy and pleurisy with effusion. Another classification is into primary or secondary forms. According to the course of the disease, a division may be made into *acute* and *chronic* pleurisy, and as it is impossible, at present, to group the various forms etiologically, this is perhaps the most satisfactory division. The following forms of acute pleurisy may be considered:

I. Fibrinous or Plastic Pleurisy.

In this the pleural membrane is covered by a sheeting of lymph of variable thickness, which gives it a turbid, granular appearance, or the fibrin may exist in distinct layers. It occurs (1) as an independent affection, following cold or exposure. This form of acute plastic pleurisy without fluid exudate is not common in perfectly healthy individuals. Cases are met with, however, in which the disease sets in with the usual symptoms of pain in the side and slight fever, and there are the physical signs of pleurisy as indicated by the friction. After persisting for a few days, the friction murmur disappears and no exudation occurs. Union takes place between the membranes, and possibly the pleuritic adhesions which are found in such a large percentage of all bodies examined after death originate in these slight fibrinous pleurisies.

Fibrinous pleurisy occurs (2) as a secondary process in acute diseases of the lung, such as pneumonia, which is always accompanied by a certain amount of pleurisy, usually of this form. Cancer, abscess, and gangrene also cause plastic pleurisy when the surface of the lung becomes involved. This condition is specially associated in a large number of cases with tuberculosis. Pleural pain, stitch in the side, and a dry cough, with marked friction sounds on auscultation are the initial phenomena in many instances of phthisis. The signs are usually basic, but Burney Yeo has recently called attention to the frequency with which they occur at the apex.

II. Sero-fibrinous Pleurisy.

In a majority of cases of inflammation of the pleura there is, with the fibrin, a variable amount of fluid exudate, which produces the condition known as pleurisy with effusion.

Etiology.—For generations physicians have considered cold the potent factor in inducing pleurisy. This may be true in many cases, but modern views of serous inflammations scarcely recognize cold as anything more than a predisposing agent, which permits the action of various micro-organisms. We have not yet, however, brought all the acute pleu-

risies into the category of microbic affections, and the fact remains that pleurisy does follow with great rapidity a sudden wetting or a chill. Of late years an attempt has been made, particularly by French writers, to show that the majority of acute pleurisies are tuberculous. In this connection the following facts may be admitted: (1) In a limited number of cases of pleurisy coming on abruptly in healthy persons the disease has been shown—(*a*) by post-mortem, in cases of accidental or sudden death, (*b*) by the subsequent history—to be tuberculous; (2) in a larger proportion of those cases which come on insidiously in persons who have been in failing health or who are delicate the disease is tuberculous from the outset; (3) the acute pleurisy, which occurs as a secondary, often a terminal, event in chronic affections, such as cirrhosis of the liver, Bright's disease, and cancer, is very frequently tuberculous. I confess that the more carefully I have studied the question the larger does the proportion appear to be of primary pleurisies of tuberculous origin. The subsequent history of cases of acute pleurisy forces us to conclude that in at least two thirds of the cases it is a curable affection. This may well be so, according to our present ideas of local tuberculous disease. One of the most interesting contributions to this question has been made from the records of Henry I. Bowditch, of Boston, to whom we are indebted for so many important contributions to our knowledge of pleurisy.* Of 90 cases of acute pleurisy which had been under observation between 1849 and 1879, 32 died of or had phthisis—a percentage large enough to indicate what an important *rôle* tuberculosis plays in the etiology of this disease.

Morbid Anatomy.—In sero-fibrinous pleurisy the serous exudate is abundant and the fibrin is found on the pleural surfaces and scattered through the fluid in the form of flocculi. The proportion of these constituents varies a great deal. In some instances there is very little membranous fibrin; in others it forms thick, creamy layers and exists in the dependent part of the fluid as whitish, curd-like masses. The fluid of sero-fibrinous pleurisy is of a citron color, either clear or slightly turbid, depending on the number of formed elements. In some instances it has a dark-brown color. The microscopical examination of the fluid shows leucocytes, occasional swollen cells, which may possibly be derived from the pleural endothelium, shreds of fibrillated fibrin, and a variable number of red blood-corpuscles. On boiling, the fluid is found to be rich in albumen. Sometimes it coagulates spontaneously. Its composition closely resembles that of blood-serum. Cholesterin, uric acid, and sugar are occasionally found. The amount of the effusion varies from a half to four litres.

The lung in acute sero-fibrinous pleurisy is more or less compressed. If the exudation is limited the lower lobe alone is atelectatic; but in an extensive effusion which reaches to the clavicle the entire lung will be found

* Vincent Y. Bowditch, in Boston Medical and Surgical Journal, 1889.

lying close to the spine, dark and airless, or even bloodless—i. e., carnified.

In large exudations the adjacent organs are displaced. In large right-sided pleurisies the liver is much depressed. Rather varying statements are made with reference to the position of the heart and as to whether or not it rotates on its axis. In a number of post-mortems I have carefully studied its position, both in pneumothorax and in large effusions, and can speak with some degree of certainty on the following points: (1) Even in the most extensive left-sided exudation there is no rotation of the apex of the heart, which in no case was to the right of the mid-sternal line; (2) the relative position of the apex and base is usually maintained; in some instances the apex is lifted, in others the whole heart lies more transversely; (3) the right chambers of the heart occupy the greater portion of the front, so that the displacement is rather a definite dislocation of the mediastinum, with the pericardium, to the right, than any special twisting of the heart itself; (4) the kink or twist in the inferior vena cava described by Bartels was not present in any of the cases.

Symptoms.—Prodromata are not uncommon, but the disease may set in abruptly with a chill, followed by fever and a severe pain in the side. It is remarkable, however, with what frequency the disease comes on insidiously. The pain in the side is the most distressing symptom, and is usually referred to the nipple or axillary regions. It must be remembered, however, that pleuritic pain may be felt in the abdomen or low down in the back, particularly when the diaphragmatic surface of the pleura is involved. It is lancinating, sharp, and severe, and is aggravated by cough. At this early stage, on auscultation, sometimes indeed on palpation, a dry friction rub can be detected. The fever rarely rises so rapidly as in pneumonia, and does not reach the same grade. A temperature of from 102° to 103° is an average pyrexia. It may drop to normal at the end of a week or ten days without the appearance of any definite change in the physical signs, or it may persist for several weeks. The temperature of the affected is higher than that of the sound side. Cough is an early symptom in acute pleurisy, but is rarely so distressing or so frequent as in pneumonia. There are instances in which it is absent. The expectoration is usually slight in amount, mucoid in character, and occasionally streaked with blood.

At the outset there may be dyspnœa, due partly to the fever and partly to the pain in the side. Later it results from the compression of the lung, particularly if the exudation has taken place rapidly. When, however, the fluid is effused slowly, one lung may be entirely compressed without inducing shortness of breath, except on exertion, and the patient will lie quietly in bed without evincing the slightest respiratory distress. When the effusion is large the patient usually prefers to lie upon the affected side.

Physical Signs.—*Inspection* shows some degree of immobility on the affected side, depending upon the amount of exudation, and in large effu-

sions an increase in volume, which may appear to be much more than it really is as determined by mensuration. The intercostal spaces are obliterated. In right-sided effusions the apex beat may be lifted to the fourth interspace or be pushed beyond the left nipple, or may even be seen in the axilla. When the exudation is on the left side the heart's impulse may not be visible; but if the effusion is large it is seen in the third and fourth spaces on the right side, and sometimes as far out as the nipple, or even beyond it.

Palpation enables us more successfully to determine the deficient movements on the affected side, and the obliteration of the intercostal spaces, and more accurately to define the position of the heart's impulse. In simple sero-fibrinous effusion there is rarely any œdema of the chest walls. It is scarcely ever possible to obtain fluctuation. Tactile fremitus is greatly diminished or abolished. If the effusion is slight there may be only enfeeblement. The absence of the voice vibrations in effusions of any size constitutes one of the most valuable of physical signs. In children there may be much effusion with retention of fremitus. In rare cases the vibrations may be communicated to the chest walls through localized pleural adhesions.

Mensuration.—With the cyrtometer, if the effusion is excessive, a difference of from half an inch to an inch, or even, in large effusions, an inch and a half, may be found between the two sides. Allowance must be made for the fact that the right side is naturally larger than the left. With the saddle-tape the difference in expansion between the two sides can be conveniently measured.

Percussion.—Early in the disease, when the pain in the side is severe and the friction murmur evident, there may be no alteration, but with the gradual accumulation of the fluid the resonance becomes defective, and finally gives place to absolute dulness. From day to day the gradual increase in height of the fluid may be studied. In a pleuritic effusion rising to the fourth rib in front, the percussion signs are usually very suggestive. In the subclavicular region the attention is often aroused at once by a tympanitic note, the so-called Skoda's resonance, which is heard perhaps more commonly in this situation with pleural effusion than in any other condition. It shades insensibly into a flat note in the lower mammary and axillary regions. Skoda's resonance may be obtained also behind, just above the limit of effusion. The dulness has a peculiarly resistant, wooden quality, differing from that of pneumonia and readily recognized by skilled fingers. It has long been known that when the patient is in the erect posture the upper line of dulness is not horizontal, but is higher behind than it is in front, forming a parabola. Ellis and Garland, of Boston, who have made a careful study of this question, state that the line of dulness from behind forward may sometimes be represented by a curved line resembling the letter S. The condition is fully considered in Garland's exhaustive work on Pneumo-dynamics.

On the right side the dulness passes without change into that of the liver. On the left side in the nipple line it extends to and may obliterate Traube's semilunar space. If the effusion is moderate, the phenomenon of movable dulness may be obtained by marking carefully, in the sitting posture, the upper limit in the mammary region, and then in the recumbent posture, noting the change in the height of dulness. This infallible sign of fluid cannot always be obtained. In very copious exudation the dulness may reach the clavicle and even extend beyond the sternal margin of the opposite side.

Auscultation.—Early in the disease a friction rub can usually be heard, which disappears as the fluid accumulates. It is a to-and-fro dry rub, close to the ear, and has a leathery, creaking character. There is another pleural friction sound which closely resembles, and is scarcely to be distinguished from, the fine crackling crepitus of pneumonia. This may be heard at the commencement of the disease, and also, as pointed out in 1844 by MacDonnell, Sr., of Montreal, when the effusion has receded and the pleural layers come together again.

With even a slight exudation there is weakened or distant breathing. Often inspiration and expiration are distinctly audible, though distant, and have a tubular quality. Sometimes only a puffing tubular expiration is heard, which may have a metallic or amphoric quality. Loud resonant râles accompanying this may forcibly suggest a cavity. These pseudo-cavernous signs are met with more frequently in children, and often lead to error in diagnosis. Above the line of dulness the breath-sounds are usually harsh and exaggerated, and may have a tubular quality.

The vocal resonance is usually diminished or absent. The whispered voice is said to be transmitted through a serous and not through a purulent exudate (Baccelli's sign). There may, however, be intensification—bronchophony. The voice sometimes has a curious nasal, squeaking character, which was termed by Laennec *ægophony*, from its supposed resemblance to the bleating of a goat. In typical form this is not common, but it is by no means rare to hear a curious twang-like quality in the voice, particularly at the outer angle of the scapula.

In the examination of the heart in cases of pleuritic effusion it is well to bear in mind that when the apex of the heart lies beneath the sternum there may be no impulse. The determination of the situation of the organ may rest with the position of maximum loudness of the sounds. In the displaced organ a systolic murmur may be heard. When the lappet of lung over the pericardium is involved on either side there may be a pleuro-pericardial friction.

The *course* of acute sero-fibrinous pleurisy is very variable. After persisting for a week or ten days the fever subsides, the cough and pain disappear, and a slight effusion may be quickly absorbed. In cases in which the effusion reaches as high as the fourth rib recovery is usually slower. Many instances come under observation for the first time, after two or

three weeks' indisposition, with the fluid at a level with the clavicle. The fever may last from ten to twenty days without exciting anxiety, though, as a rule, in ordinary pleurisy from cold, as we say, the temperature in cases of moderate severity is normal within eight or ten days. Left to itself the natural tendency is to resorption; but this may take place very slowly. Even after it has persisted for months a sero-fibrinous exudate may completely disappear. With the absorption of the fluid there is a redux-friction crepitus, either leathery and creaking or crackling and râle-like, and for months, or even longer, the defective resonance and feeble breathing are heard at the base.

A sero-fibrinous exudate may persist for months without change, particularly in tuberculous cases, and will sometimes reaccumulate after aspiration and resist all treatment. The change of the exudate into pus will be spoken of in connection with empyema. Death is a rare termination of sero-fibrinous effusion. When one pleura is full and the heart is greatly dislocated the condition, although in a majority of cases producing remarkably little disturbance, is not without risk. *Sudden death* may occur, and its possibility under these circumstances should always be considered. I have seen two instances—one in right and the other in left sided effusion—both due, apparently, to syncope following slight exertion, such as getting out of bed. In neither case, however, was the amount of fluid excessive. Weil, who has studied carefully this accident, concludes as follows: (1) That it may be due to thrombosis or embolism of the heart or pulmonary artery, œdema of the opposite lung, or degeneration of the heart muscle; (2) such alleged causes as mechanical impediment to the circulation, owing to dislocation of the heart or twisting of the great vessels, require further investigation. It occurs more frequently in right than in left pleurisies, and the effusion is usually serous. Death may occur without any premonitory symptoms, usually during some movement or effort.

III. Purulent Pleurisy (*Empyema*).

Etiology.—Pus in the pleura is met with under the following conditions: (*a*) As a sequence of acute sero-fibrinous pleurisy. It is not always easy to say why, in certain cases, the exudate becomes purulent. It rarely does so in the acute pleurisies of healthy individuals. In children many cases are probably purulent from the outset. Aspiration, which is said to favor the occurrence of empyema, in my experience does so very rarely. (*b*) Purulent pleurisy is common as a secondary inflammation in various infectious diseases, among which scarlet fever takes the first place. It has long been known that the pleurisy supervening in the convalescence of this disease is almost always purulent. It should be remembered that it is latent in its onset, and that there may be no pulmonary symptoms. The pleurisy following typhoid fever is also usually purulent. Other infectious diseases—measles and whooping-cough

—are more rarely followed by this complication. Of late years especial attention has been paid to the connection of pneumonia with empyema, and it has been shown that very many cases come on insidiously either in the course of or during convalescence from this disease; and, lastly, a limited number of tuberculous pleurisies early become purulent. (*c*) Empyema results from local causes—fracture of the rib, penetrating wounds, malignant disease of the lung or œsophagus, and, perhaps most frequently of all, the perforation of the pleura by tuberculous cavities.

The bacteriology of empyema is of some importance. A sterile exudate suggests tuberculosis. In many cases the pneumococci are present, and these, as a rule, run a very favorable course. The streptococci are found most commonly in the secondary cases in connection with septic processes. In a few instances psorosperms have been found in the exudate.

Morbid Anatomy.—On opening an empyema post mortem, we usually find that the effusion has separated into a clear, greenish-yellow serum above and the thick, cream-like pus below. The fluid may be scarcely more than turbid, with flocculi of fibrin through it. In other instances it is uniformly thick and creamy, without any fibrin. It usually has a heavy, sweetish odor, but in some instances—particularly those following wounds—it is fetid. In cases of gangrene of the lung or pleura the pus has a horribly stinking odor. Microscopically it has the characters of ordinary pus. The pleural membranes are greatly thickened, and present a grayish-white layer from 1 to 2 mm. in thickness. On the costal pleura there may be erosions, and in old cases fistulous communications are common. The lung may be compressed to a very small limit, and the visceral pleura also may show perforations.

Symptoms.—Purulent pleurisy may begin abruptly, with the symptoms already described. More frequently it comes on insidiously in the course of other diseases or follows an ordinary sero-fibrinous pleurisy. There may be no pain in the chest, very little cough, and no dyspnœa, unless the side is very full. Symptoms of septic infection are rarely wanting. If in a child, there is a gradually developing pallor and weakness; sweats occur, and there is irregular fever. A cough is by no means constant.

Physical Signs.—Practically they are those already considered in pleurisy with effusion. There are, however, one or two additional points to be mentioned. In empyema, particularly in children, the disproportion between the sides may be extreme. The intercostal spaces may not only be obliterated, but may bulge. Much more frequently there is œdema of the chest walls. The network of subcutaneous veins may be very distinct. It must not be forgotten that in children the breath-sounds may be *loud and tubular* over a purulent effusion of considerable size. Whispered pectoriloquy is usually not heard in empyema (Baccelli's sign). The dislocation of the heart and the displacement of the liver are more marked

in empyema than in sero-fibrinous effusion—probably, as Senator suggests, owing to the greater weight of the fluid.

A curious phenomenon associated generally with empyema, but which may occur in the sero-fibrinous exudate, is *pulsating pleurisy*, first described by MacDonnell, Sr., of Montreal. Of 42 cases 39 occurred on the left side. In all but one case the fluid was purulent. Pneumothorax may be present. There are two groups of cases, the intrapleural pulsating pleurisy and the pulsating *empyema necessitatis*, in which there is an external pulsating tumor. No satisfactory explanation has been offered how the heart impulse is thus forcibly communicated through the effusion.

Empyema is a chronic affection, which in a few instances terminates naturally in recovery, but a majority of cases, if left alone, end in death. The following are some modes of natural cure: (*a*) By absorption of the fluid. In small effusions this may take place gradually. The chest wall sinks. The pleural layers become greatly thickened and enclose between them the inspissated pus, in which lime salts are gradually deposited. Such a condition may be seen once or twice a year in the post-mortem room of any large hospital. (*b*) By perforation of the lung. Although in this event death may take place rapidly, by inundation of the bronchial tubes, yet in many cases it occurs gradually and recovery follows. Since 1873, when I saw a case of this kind in Traube's clinic, and heard his remarks on the subject, I have seen a number of instances of the kind and can corroborate his statement as to the favorable termination of many of them. Empyema may discharge either by opening into the bronchus and forming a fistula or, as Traube pointed out, by producing necrosis of the pulmonary pleura, sufficient to allow the soakage of the pus through the spongy lung tissue into the bronchi. In the first way pneumothorax usually, though not always, develops. In the second way the pus is discharged without formation of pneumothorax. Even with a bronchial fistula recovery is possible. (*c*) By perforation of the chest wall—*empyema necessitatis*. This is by no means an unfavorable method, as many cases recover. The perforation may occur anywhere in the chest wall, but is, as Cruveilhier remarked, more common in front. It may be anywhere from the third to the sixth interspace, usually, according to Marshall, in the fifth. It may perforate in more than one place, and there may be a fistulous communication which opens into the pleura at some distance from the external orifice. The tumor, when near the heart, may pulsate. The discharge may persist for years. In Copeland's Dictionary is mentioned an instance of a Bavarian physician who had a pleural fistula for thirteen years and enjoyed fairly good health.

An empyema may perforate the neighboring organs, the œsophagus, peritonæum, pericardium, or the stomach. Very remarkable cases are those which pass down the spine and along the psoas into the iliac fossa, and simulate a psoas or lumbar abscess.

IV. Tuberculous Pleurisy.

This has already been considered. Here it is sufficient to say that it occurs as: (*a*) An acute affection, accompanied by abundant sero-fibrinous fluid. In this category come certainly a proportion of the cases regarded as acute pleurisy from cold. (*b*) As a subacute affection, latent in its origin and insidious in its course, frequently preceding the development of or coming on concurrently with pulmonary tuberculosis. (*c*) As an acute pleurisy, the result of direct extension from the lung in cases of well-marked phthisis, and in which the fluid may be either sero-fibrinous or purulent. (*d*) Chronic adhesive tuberculous pleurisy, which may be unilateral or bilateral, unaccompanied by exudation and characterized by great thickening of the pleural membranes, in which are tubercles and caseous masses of varying sizes.

The symptoms and physical signs of tuberculous pleurisy with exudation do not require any description other than that already given in connection with the sero-fibrinous and purulent forms.

V. Other Varieties of Pleurisy.

Hæmorrhagic Pleurisy.—A bloody effusion is met with under the following conditions: (*a*) In the pleurisy of asthenic states, such as cancer, Bright's disease, and occasionally in the malignant fevers. It is interesting to note the frequency with which hæmorrhagic pleurisy is found in cirrhosis of the liver. It occurred in the very patient in whom Laennec first accurately described this disease. While this may be a simple hæmorrhagic pleurisy, in a majority of the cases which I have seen it has been tuberculous. (*b*) Tuberculous pleurisy, in which the bloody effusion may result from the rupture of newly formed vessels in the soft exudate accompanying the eruption of miliary tubercles, or it may come from more slowly formed tubercles in a pleurisy secondary to extensive pulmonary disease. (*c*) Cancerous pleurisy, whether primary or secondary, is frequently hæmorrhagic. (*d*) Occasionally hæmorrhagic exudation is met with in perfectly healthy individuals, in whom there is not the slightest suspicion of tuberculosis or cancer. In one such case, a large, able-bodied man, the patient was to my knowledge healthy and strong eight years afterward. And, lastly, it must be remembered that during aspiration the lung may be wounded and blood in this way get mixed with the sero-fibrinous exudate. The condition of hæmorrhagic pleurisy is to be distinguished from hæmothorax, due to the rupture of aneurism or the pressure of a tumor on the thoracic veins.

Diaphragmatic Pleurisy.—The inflammation may be limited partly or chiefly to the diaphragmatic surface. This is often a dry pleurisy, but there may be effusion, either sero-fibrinous or purulent, which is circumscribed on the diaphragmatic surface. In these cases the pain is low in

the zone of the diaphragm and, as Guéneau de Mussy pointed out, may be intensified by pressure at the point of insertion of the diaphragm at the tenth rib. The diaphragm is fixed and the respiration is thoracic and short. Andral noted in certain cases severe dyspnœa and attacks simulating angina. As mentioned, the effusion is usually plastic, not serous. Serous or purulent effusions of any size limited to the diaphragmatic surface are extremely rare.

Encysted Pleurisy.—The effusion may be circumscribed by adhesions or separated into two or more pockets or loculi, which communicate with each other. This is most common in empyema. In these cases there have usually been, at different parts of the pleura, multiple adhesions by which the fluid is limited. In other instances the recent false membranes may encapsulate the exudation on the diaphragmatic surface, for example, or the part of the pleura posterior to the mid-axillary line. The condition may be very puzzling during life, and present special difficulties in diagnosis. In some cases the tactile fremitus is retained along certain lines of adhesion. The exploratory needle should be freely used when there is any doubt.

Interlobar Pleurisy forms an interesting and not uncommon variety. In nearly every instance of acute pleurisy the interlobular serous surfaces are also involved and closely agglutinated together, and sometimes the fluid is encysted between them. In a recent case of this kind following pneumonia, there was between the lower and upper and middle lobes of the right side an enormous purulent collection, which looked at first like a large abscess of the lung. These collections may perforate the bronchi, and the cases present special difficulties in diagnosis.

Diagnosis of Pleurisy.—Acute plastic pleurisy is readily recognized. In the diagnosis of pleuritic effusion the first question is, Does a fluid exudate exist? the second, What is its nature? In large effusions the increase in the size of the affected side, the immobility, the absence of tactile fremitus, together with the displacement of organs, give infallible indications of the presence of fluid. The chief difficulty arises in effusions of moderate extent, when the dulness, the presence of bronchophony, and, perhaps, tubular breathing may simulate *pneumonia*. The chief points to be borne in mind are: (*a*) Differences in the onset and in the general characters of the two affections, more particularly the initial chill, the higher fever, more urgent dyspnœa, and the rusty expectoration, which characterize pneumonia. (*b*) Certain physical signs—the more wooden character of the dulness, the greater resistance, and the marked diminution or the absence of tactile fremitus in pleurisy. The auscultatory signs may be deceptive. It is usually, indeed, the persistence of tubular breathing, particularly the high-pitched, even amphoric expiration, heard in some cases of pleurisy, which has raised the doubt. The intercostal spaces are more commonly obliterated in pleuritic effusion than in pneumonia. As already mentioned, the displacement of organs is a very valuable sign.

Nowadays with the hypodermic needle the question is easily settled. A separate small syringe with a capacity of two drachms should be reserved for exploratory purposes, and the needle should be longer and firmer than in the ordinary hypodermic instrument. With careful preliminary disinfection the instrument can be used with impunity, and in cases of doubt the exploratory puncture should be made without hesitation. I have never seen the slightest ill effects follow its use. Cases are reported of pneumothorax resulting from it, but they are extremely rare. The hypodermic needle is especially useful in those cases in which there are pseudo-cavernous signs at the base. In cases, too, of massive pneumonia, in which the bronchi are plugged with fibrin, if the patient has not been seen from the outset, the diagnosis may be impossible without it.

On the left side it may be difficult to differentiate a very large pericardial from a pleural effusion. The retention of resonance at the base, the presence of Skoda's resonance toward the axilla, the absence of dislocation of the heart-beat to the right of the sternum, the feebleness of the pulse and of the heart-sounds, and the urgency of the dyspnœa, out of all proportion to the extent of the effusion, are the chief points to be considered. Unilateral hydrothorax, which is not at all uncommon in heart-disease, presents signs identical with those of sero-fibrinous effusion. Certain tumors within the chest may simulate pleural effusion. It should be remembered that many intrathoracic growths are accompanied by exudation. Malignant disease of the lung and of the pleura and hydatids of the pleura produce extensive dulness, with suppression of the breath-sounds, simulating closely effusion.

On the right side abscess of the liver and hydatid cysts may rise high into the pleura and produce dulness and enfeebled breathing. Often in these cases there is a friction sound, which should excite suspicion, and the upper outline of the dulness is sometimes plainly convex. In all these instances the exploratory puncture should be made.

The second question, as to the nature of the fluid, is quickly decided by the use of the needle. The persistent fever, the occurrence of sweats, and the increase in the pallor suggest the presence of pus. In children the complexion is often sallow and earthy. The unexpected, however, often happens, and repeatedly, in protracted cases, even in children, when the general symptoms and the appearance of the patient has been most strongly suggestive of pus, the syringe has withdrawn clear fluid. On the other hand, effusions of short duration may be purulent, even when the general symptoms do not suggest it. The following statement may be made with reference to the prognostic import of the bacteriological examination of the aspirated fluid: The presence of the pneumococcus is of favorable significance, as such cases usually get well rapidly, even with a single aspiration. The pus organisms—staphylococci and streptococci—are more common in empyema of septic origin, and such cases are notori-

ously less hopeful than others. A sterile fluid indicates in a majority of instances a tuberculous origin.

Treatment.—At the onset the severe pain may demand leeches, which usually give relief, but a hypodermic of morphia is more effective. The Paquelin cautery may be lightly but freely applied. It is well to administer a mercurial or saline purge. Fixing the side by careful strapping with long strips of adhesive plaster, which should pass well over the middle line, drawn tightly and evenly, gives great relief, and I can corroborate the statement of F T. Roberts as to its efficacy. Cupping, wet or dry, is now seldom employed. Blisters are of no special service in the acute stages, although they relieve the pain. The ice-bag may be used as in pneumonia. The general treatment of the early stage should be rest in bed and a liquid diet. Medicines are rarely required. A Dover's powder may be given at night. Mercurials are not indicated.

When the effusion has taken place, mustard plasters or iodine, producing slight counter-irritation, appear useful, particularly in the later stages. The following rational plan is successful in some cases. It is based upon the idea that if the blood serum is depleted or if it is kept concentrated, the liquid will be absorbed from the lymph spaces, of which the pleura is one, to equalize the loss. To do this the patient should have the daily amount of liquid food greatly restricted. If there is no fever, a meat diet, with an egg and dry bread and eight to ten ounces of liquid in the form of milk or water, should be given. Salt articles of food may be used, but I do not think it necessary to give, as some do, doses of salt. The second element in the treatment is the active depletion of blood serum, which is effected in the way introduced by Matthew Hay. Every morning, if the patient is robust, otherwise every second morning, from half an ounce to an ounce and a half of Epsom salts is given an hour before breakfast, in as concentrated a form as is possible. This produces copious liquid discharges. I have seen large exudations disappear rapidly when this plan was followed. By acting upon the skin and kidneys, the same end may be obtained, but with much less certainty. The vapor or hot bath may be used and an occasional dose of pilocarpin. Diuretics, such as digitalis, squills, and acetate of potash, may sometimes be required. I rarely resort, however, to diuretics or diaphoretics in the treatment of pleurisy with effusion. Iodide of potassium is of doubtful benefit.

Aspiration of the fluid is the most thorough and satisfactory method and should be resorted to whenever the effusion becomes large or if it resists the ordinary methods of treatment. The credit of introducing aspiration in pleuritic effusions is due to Morrill Wyman, of Cambridge, Mass., and Henry I. Bowditch, of Boston. Years prior to Dieulafoy's work, aspiration was in constant use at the Massachusetts General Hospital and was advocated repeatedly by Bowditch. As the question is one of some historical interest, I give the author's conclusions concerning aspiration, expressed more than forty years ago, and which practically represent

the opinion of clinical physicians to-day: "(1) The operation is perfectly simple, but slightly painful, and can be done with ease upon any patient in however advanced a stage of the disease. (2) It should be performed forthwith in *all* cases in which there is complete filling up of one side of the chest. (3) He had determined to use it in *any* case of even *moderate* effusion lasting more than a few weeks and in which there should seem to be an indisposition to resist ordinary modes of treatment. (4) He urged this practice upon the profession as a very important measure in practical medicine; believing that by this method death may frequently be prevented from ensuing either by sudden attack of dyspnœa or subsequent phthisis, and, finally, from the gradual wearing out of the powers of life or inability to absorb the fluid. (5) He believed that this operation would sometimes prevent the occurrence of those tedious cases of spontaneous evacuation of purulent fluid and those great contractions of the chest which occur after long-continued effusion and the subsequent discharge or absorption of a fluid."

There is scarcely anything to be added to-day to these observations. When the fluid reaches to the clavicle the indication for aspiration is imperative, even though the patient be comfortable and present no signs of pulmonary distress. The presence of fever is not a contra-indication; indeed, sometimes with serous exudates the temperature falls after aspiration.

The operation is extremely simple and is practically without risk. The spot selected for puncture should be either in the seventh interspace in the mid-axilla or at the outer angle of the scapula in the eighth interspace. The arm of the patient should be brought forward with the hand on the opposite shoulder, so as to widen the interspaces. The needle should be thrust in close to the upper margin of the rib, so as to avoid the intercostal artery, the wounding of which, however, is an excessively rare accident. The fluid should be withdrawn slowly. The amount will depend on the size of the exudate. If the fluid reaches to the clavicle a litre or more may be withdrawn with safety.

During aspiration if the patient feels faint it is best to interrupt the operation, for sudden death has occasionally happened during the withdrawal. It is, however, a much less common accident than sudden death in cases of full pleura without operation. Cough is a symptom which frequently develops toward the close of aspiration. Though very painful it need not excite alarm. French writers have described cases of albuminous expectoration, associated with dyspnœa, which may come on after the tapping and prove rapidly fatal. It must be an excessively rare complication. The conversion of a sero-fibrinous into a purulent fluid is a danger which need not be considered. I have never met with an instance of the kind.

Empyema is really a surgical affection, and I shall make only a few general remarks upon its treatment. When it has been determined by

exploratory puncture that the fluid is purulent, aspiration should not be performed, except as preliminary to operation or as a temporary measure. Perhaps it is better not to have an exception to this rule, although the empyemas of children and the pneumonic empyema occasionally get well rapidly after a single tapping. It is sad to think of the number of lives which are sacrificed annually by the failure to recognize that empyema should be treated as an ordinary abscess, by free incision. The operation dates from the time of Hippocrates and is by no means serious. A majority of the cases get well, providing that free drainage is obtained, and it makes no difference practically what measures are followed so long as this indication is met. The good results in any method depend upon the thoroughness with which the cavity is drained. Irrigation of the cavity is rarely necessary unless the contents are fetid. Sudden collapse has happened during irrigation and a remarkable accident is the occurrence of convulsions. In the subsequent treatment a point of great importance in facilitating the closure of the cavity is the distention of the lung on the affected side. This may be accomplished by the method advised by Walter James, which has been practised with great success in the surgical wards of the Johns Hopkins Hospital. The patient daily, for a certain length of time, increasing gradually with the increase of his strength, transfers by air-pressure water from one bottle to another. The bottles should be large, holding at least a gallon each, and by the arrangement of tubes, as in the Wolff's bottle, an expiratory effort of the patient forces the water from one bottle into the other. In this way expansion of the compressed lung is systematically practised. The abscess cavity is gradually closed, partly by the falling in of the chest wall and partly by the expansion of the lung. In some instances it is necessary to resect portions of one or more ribs.

The physician is often asked, in cases of empyema with emaciation, hectic and feeble rapid pulse, whether the patient could stand the operation. Even in the most desperate cases the surgeon should never hesitate to make a free incision.

II. CHRONIC PLEURISY.

This affection occurs in two forms: (1) *Chronic pleurisy with effusion*, in which the disease may set in insidiously or may follow an acute serofibrinous pleurisy. There are cases in which the liquid persists for months without undergoing any special alteration and without becoming purulent. Such cases have the characters which we have described under pleurisy with effusion. (2) *Chronic dry pleurisy*. The cases are met with (*a*) as a sequence of ordinary pleural effusion. When the exudate is absorbed and the layers of the pleura come together there is left between them a variable amount of fibrinous material which gradually undergoes organi-

zation, and is converted into a layer of firm connective tissue. This process goes on at the base, and is represented clinically by a slight grade of flattening, deficient expansion, defective resonance on percussion, and enfeebled breathing. After recovery from empyema the flattening and retraction may be still more marked. In both cases it is a condition which can be greatly benefited by pulmonary gymnastics. In these firm, fibrous membranes calcification may occur, particularly after empyema. It is not very uncommon to find between the false membranes a small pocket of fluid forming a sort of pleural cyst. In the great majority of these cases the condition is one which need not cause anxiety. There may be an occasional dragging pain at the base of the lung or a stitch in the side, but patients may remain in perfectly good health for years. The most advanced grade of this secondary dry pleurisy is seen in those cases of empyema which have been left to themselves and have perforated and ultimately healed by a gradual absorption or discharge of the pus, with retraction of the side of the chest and permanent carnification of the lung. Traumatic lesions, such as gunshot wounds, may be followed by an identical condition. Post mortem, it is quite impossible to separate the layers of the pleura, which are greatly thickened, particularly at the base, and surround a compressed, airless, fibroid lung.

(*b*) *Primitive dry pleurisy.* This condition may directly follow the acute plastic pleurisy already described; but it may set in without any acute symptoms whatever, and the patient's attention may be called to it by feeling the pleural friction. A constant effect of this primitive dry pleurisy is the adhesion of the layers. This is probably an invariable result, whether the pleurisy is primary or secondary. The organization of the thin layer of exudation in a pneumonia will unite the two surfaces by delicate bands. Pleural adhesions are extremely common, and it is rare to examine a body entirely free from them. They may be limited in extent or universal. Thin fibrous adhesions do not produce any alteration in the percussion characters, and, if limited, there is no special change heard on auscultation. When, however, there is general synechia on both sides the expansile movement of the lung is considerably impaired. We should naturally think that universal adhesions would interfere materially with the function of the lungs, but practically we see many instances in which there has not been the slightest disturbance. The physical signs of total adhesion are by no means constant. It has been stated that there is a marked disproportion between the degree of expansion of the chest walls and the intensity of the vesicular murmur, but the latter is a very variable factor, and under perfectly normal conditions the breath-sounds, with very full chest expansion, may be extremely feeble.

Is there a primitive dry pleurisy which gradually leads to great thickening of the membranes, and which ultimately may invade the lung and induce cirrhotic change? Upon this question neither pathologists nor clinicians agree. I think that Sir Andrew Clark, in his Lumleian lectures

at the Royal College of Physicians (1885), has made good his claim that such a disease does exist. At the outset in these cases there is a dry pleurisy, usually at one base, indicated by the usual signs; and this persists in spite of all treatment. There is no evidence of fluid; the general health may not be much impaired, or there may be slight fever and disturbed digestion. The cases give great anxiety, owing to the natural suspicion that tuberculosis exists. In time the evidence of dulness is found at the base. There are feeble breathing and creaking, leathery friction sounds. There may be commencing retraction of the side. Clinically these cases are of great interest, and should, I think, be separated, on the one hand, from the condition which follows a healed empyema or old pleurisy with effusion, and, on the other, from the rare instances of primitive cirrhosis of the lung. However, in all three states there may ultimately be an almost identical clinical picture. Anatomically in these pleuritic cases the pleura, particularly that surrounding the lower lobe, sometimes the entire membrane, is thickened, the two layers are intimately united, and fibrinous bands passing from the pleura traverse the lung tissue, sometimes dividing it in a remarkable way into sections. The bronchi may present marked dilatations, though this is not always the case, and the lung tissue is more or less sclerosed. The cases belong to the group of chronic pneumonias called by Charcot pleurogenous. In many instances there can be no question as to their non-tuberculous nature. There are cases, however, in which, with chronic pleurogenous pneumonia in the lower lobe, there are cavity formations at the apex and tuberculous lesions in other parts. Such may, of course, be tuberculous from the outset.

Lastly, there is a primitive dry pleurisy of tuberculous origin. In it both parietal and costal layers are greatly thickened—perhaps from two to three millimetres each—and present firm fibroid, caseous masses and small tubercles, while uniting these two greatly thickened layers is a reddish-gray fibroid tissue, sometimes infiltrated with serum. This may be a local process confined to one pleura, or it may be in both. I have seen two typical instances of it—one in a young, well-nourished Irish girl, who died of malignant scarlet fever, in whom one pleura was in the condition above described, and there were no other tuberculous lesions. The other was in a young man who died of typhoid fever, in whom both pleuræ were uniformly thickened and tuberculous without any fluid exudate. These cases are sometimes associated with a similar condition of the pericardium and peritonæum.

Occasionally remarkable vaso-motor phenomena occur in chronic pleurisy, whether simple or in connection with tuberculosis of an apex. Flushing or sweating of one cheek or dilatation of the pupil are the common manifestations. They appear to be due to involvement of the first thoracic ganglion at the top of the pleural cavity.

III. HYDROTHORAX.

Hydrothorax is a transudation of simple non-inflammatory fluid into the pleural cavities, and occurs as a secondary process in many affections. The fluid is clear, without any flocculi of fibrin, and the membranes are smooth. It is met with more particularly in connection with general dropsy, either renal, cardiac, or hæmic. It may, however, occur alone, or with only slight œdema of the feet. A child was admitted to the Montreal General Hospital with urgent dyspnœa and cyanosis, and died the night after admission. She had extensive bilateral hydrothorax, which had come on early in the nephritis of scarlet fever. In renal disease hydrothorax is almost always bilateral, but in heart affections one pleura is more commonly involved. The physical signs are those of pleural effusion, but the exudation is rarely excessive. In kidney and heart disease, even when there is no general dropsy, the occurrence of dyspnœa should at once direct attention to the pleura, since many patients are carried off by a rapid effusion. Post-mortem records show the frequency with which this condition is overlooked. The saline purges will in many cases rapidly reduce the effusion, but, if necessary, aspiration should repeatedly be practised.

IV. PNEUMOTHORAX (*Hydro-Pneumothorax and Pyo-Pneumothorax*).

Air alone in the pleural cavity, to which the term pneumothorax is strictly applicable, is an extremely rare condition. It is almost invariably associated with a serous fluid—hydro-pneumothorax, or with pus—pyo-pneumothorax.

Etiology.—It has usually been taught that there is an inherent tendency to pneumothorax, which is induced as soon as the pleura is opened. The experiments of S. West seem, however, to indicate the existence of a coherent force between the pleural surfaces much in excess of the elasticity of the lung, and sufficient in certain instances to maintain these organs in contact with the thoracic wall, even when there is free access to the pleura; so that in reality force is required to overcome the normal adhesion between the pleural membranes.

Pneumothorax arises: (1) In perforative wounds of the chest, in which case it is sometimes associated with extensive cutaneous emphysema. It has followed exploratory puncture with a hypodermic needle, as in two cases reported by Herman Biggs. Pneumothorax rarely follows fracture of the rib, even though the lung may be torn. (2) In perforation of the pleura through the diaphragm, usually by malignant disease of the stomach or colon. The pleura may also be perforated in cases of cancer of the œsophagus. (3) When the lung is perforated. This is by far the most common cause, and may occur: (*a*) In a normal lung from rupture

of the air-vesicles during straining. Special attention has lately been called to this accident by S. West and De H. Hall. The air may be absorbed and no ill effect follows. It does not necessarily excite pleurisy, as pointed out many years ago by Gairdner, but inflammation and effusion are the usual result. (*b*) From perforation due to local disease of the lung, either the softening of a caseous focus or the breaking of a tuberculous cavity. According to S. West, ninety per cent of all the cases are due to this cause. Less common are the cases due to septic bronchopneumonia and to gangrene. A rare cause is the breaking of a hæmorrhagic infarct in chronic heart-disease, of which I met an instance a few years ago. (*c*) Perforation of the lung from the pleura, which arises in certain cases of empyema and produces a pleuro-bronchial fistula.

Pneumothorax occurs chiefly in adults, though cases are met with in very young children. It is more frequent in males than in females.

Morbid Anatomy.—If a trocar or blow-pipe is inserted between the ribs, there may be a jet of air of sufficient strength to blow out a lighted match. On opening the thorax the mediastinum and pericardium are seen to be pushed, or rather, as Douglas Powell pointed out, drawn over to the opposite side; but, as before mentioned, the heart is not rotated, and the relation of its parts is maintained much as in the normal condition. A serous or purulent fluid is usually present, and the membranes are inflamed. The cause of the pneumothorax can usually be found without difficulty. In the great majority of instances it is the perforation of a tuberculous cavity or a breaking of a superficial caseous focus. The orifice of rupture may be extremely small. In chronic cases there may be a fistula of considerable size communicating with the bronchi. The lung is usually compressed and carnified.

Symptoms.—The onset is usually sudden and characterized by severe pain in the side, urgent dyspnœa, and signs of general distress, as indicated by slight lividity and a very rapid and feeble pulse. There may, however, be no urgent symptoms, particularly in cases of long-standing phthisis. On more than one occasion I have found, post mortem, a pneumothorax which was unsuspected during life. West states that even in healthy adults this latent pneumothorax may occasionally occur.

The *physical signs* are very distinctive. *Inspection* shows marked enlargement of the affected side with immobility. The heart impulse is usually much displaced. On *palpation* the fremitus is greatly diminished or more commonly abolished. On *percussion* the resonance may be tympanitic or even have an amphoric quality. This, however, is not always the case. It may be a flat tympany, resembling Skoda's resonance. In some instances it may be a full, hyperresonant note, like emphysema; while in others—and this is very deceptive—there is dulness. These extreme variations depend doubtless upon the degree of intrapleural tension. On several occasions I have known an error in diagnosis to result from ignorance of the fact that, in certain instances, the percussion note

may be "muffled, toneless, almost dull" (Walshe). There is usually dulness at the base from effused fluid, which can readily be made to change the level by altering the position of the patient. Movable dulness can be obtained much more readily in pneumothorax than in a simple pleurisy. On *auscultation* the breath-sounds are suppressed. Sometimes there is only a distant feeble inspiratory murmur of marked amphoric quality. The contrast between the loud exaggerated breath-sounds on the normal side and the absence of the breath-sounds on the other is very suggestive. The râles have a peculiar metallic quality, and on coughing or deep inspiration there may be what Laennec termed the metallic tinkling. The voice, too, has a curious metallic echo. What is sometimes called the coin-sound, termed by Trousseau the *bruit d'airain*, is very characteristic. To obtain it the auscultator should place one ear on the back of the chest wall while the assistant taps one coin on another on the front of the chest. The metallic echoing sound which is produced in this way is one of the most constant and characteristic signs of pneumothorax. And, lastly, the Hippocratic succussion may be obtained when the auscultator's head is placed upon the patient's chest and his body shaken. A splashing sound is produced, which may be audible at a distance. A patient may himself notice it in making abrupt changes in posture. Of other symptoms displacement of organs is most constant. As already mentioned, the heart may be drawn over to the opposite side, and the liver greatly displaced, so that its upper surface is below the level of the costal margin, a degree of dislocation never seen in simple effusion.

The *diagnosis* of pneumothorax rarely offers any difficulty, as the signs are very characteristic. In cases in which the percussion note is dull the condition may be mistaken for effusion. I made this mistake in a case of pulsating pleurisy, in which the pneumothorax followed heavy lifting, and it was not until several days later, after some of the fluid had been withdrawn, that a tympanitic note developed. Diaphragmatic hernia following a crush or other accident may closely simulate pneumothorax.

In cases of very large phthisical cavities with tympanitic percussion resonance and râles of an amphoric, metallic quality the question of pneumothorax is sometimes raised. In those rare instances of total excavation of one lung the amphoric and metallic phenomena may be most intense, but the absence of dislocation of the organs and of the succussion splash and of the coin sound suffice to differentiate this condition. Why the coin sound is not heard it is difficult to determine, unless its production is connected in some way with a certain degree of air-tension, which is not present in a vomica, however large. The condition of pyopneumothorax subphrenicus may simulate closely true pneumothorax.

The *prognosis* in cases of pneumothorax depends largely upon the cause. The phthisical cases usually die within a few weeks. Pneumothorax developing in a healthy individual often ends in recovery. There are cases of phthisis in which the pneumothorax, if occurring early, seems

to arrest the progress of the tuberculosis. This appeared to be the case in a man with chronic pneumothorax who was under my care in Philadelphia for between three and four years. It may be a chronic condition, as in the case just mentioned, and a fair measure of health may be enjoyed.

Treatment.—Practically these cases should be dealt with as ordinary pleurisy with effusion. Of course, when pneumothorax develops in advanced phthisis the indication is to relieve the pain and distress either by morphia or chloroform; but in cases which develop early the fluid should be withdrawn by aspiration, or, if purulent, permanent drainage should be obtained. Even when the condition has seemed to be most desperate I have known recovery to take place after thorough drainage of the sac. Portions of ribs may have to be excised, and during convalescence it is well for the patient to practise expansion of the lung in the manner already mentioned. There are cases of pneumothorax in phthisis in which the general condition is so good and the inconvenience so slight that to let well enough alone seems the best course. In such an occasional aspiration may be performed if the fluid increases. In some of the instances the mere tapping of the chest with a fine needle, so as to allow the escape of some of the air, seems to give relief by reducing the intrathoracic pressure. Good results are stated to have followed the method introduced by Potain, of replacing the air and fluid within the thorax by sterilized air.

AFFECTIONS OF THE MEDIASTINUM.

(1) **Simple Lymphadenitis.**—In all inflammatory affections of the bronchi and of the lungs the groups of lymph glands in the mediastinum become swollen. In the bronchitis of measles, for example, and in simple broncho-pneumonia the bronchial glands are large and infiltrated, the tissue is engorged and œdematous, sometimes intensely hyperæmic. Much stress has been laid by some writers on this enlargement of the glands in the posterior mediastinum, and De Mussy held that it was an important factor in inducing paroxysms of whooping-cough. They may attain a size sufficient to induce dulness beneath the manubrium and in the upper part of the interscapular regions behind, though this is often difficult to determine. In reality the glands lie chiefly upon the spine, and unless those which are deep in the root of the lung are large enough to induce compression of the adjacent lung tissue, I doubt if the ordinary bronchial adenopathy ever can be determined by percussion in the upper interscapular region. I have never met with an instance in which the compression of either bronchus seemed to have resulted from the glands, however large. Tuberculous affection of these glands has already been considered.

(2) **Suppurative Lymphadenitis.**—Occasionally abscess in the bronchial or tracheal lymph glands is found. It may follow the simple adenitis, but

is most frequently associated with the presence of tubercle. The liquid portion may gradually become absorbed and the inspissated contents undergo calcification. Serious accident occasionally occurs, as perforation into the œsophagus or into a bronchus.

(3) **Tumors; Cancer and Sarcoma.**—In Hare's elaborate study of 520 cases of disease of the mediastinum* there were 134 cases of cancer, 98 cases of sarcoma, 21 cases of lymphoma, 7 cases of fibroma, 11 cases of dermoid cysts, 8 cases of hydatid cysts, and instances of lipoma, gumma, and enchondroma. From this we see that cancer is the most common form of growth. The tumor occurred in the anterior mediastinum alone in 48 of the cases of cancer and 33 of the cases of sarcoma. The disease may be either primary in the mediastinal tissues and lymph structures or secondary. Sarcoma is more frequently primary than cancer. Males are more frequently affected than females. The age of onset is most commonly between thirty and forty.

Symptoms.—The signs of mediastinal tumor are those of intrathoracic pressure. *Dyspnœa* is one of the earliest and most constant symptoms, and may be due either to pressure on the trachea or on the recurrent laryngeal nerves. It may indeed be cardiac, due to pressure upon the heart or its vessels. In a few cases it results from the pleural effusion which so frequently accompanies intrathoracic growths. Associated with the dyspnœa is a cough, often severe and paroxysmal in character, with the brazen quality of the so-called aneurismal cough when a recurrent nerve is involved. The voice may also be affected from a similar cause. Pressure on the vessels is common. The superior vena cava may be compressed and obliterated, and when the process goes on slowly the collateral circulation may be completely effected. Less commonly the inferior vena cava or one or other of the subclavian veins is compressed. The arteries are much less rarely obstructed. It is remarkable how little the aorta may be involved, though entirely surrounded by a sarcomatous or cancerous mass. There may be dysphagia, due to compression of the œsophagus. In rare instances there are pupillary changes, either dilatation or contraction, due to involvement of the sympathetic.

Physical Signs.—On inspection there may be orthopnœa and marked cyanosis of the upper part of the body. In such instances, if of long duration, there are signs of collateral circulation and the superficial mammary and epigastric veins are enlarged. In a patient with Hodgkin's disease, at present under observation and in whom during the past sixteen months there has been progressive compression and now obliteration of the superior vena cava, the entire subcutaneous tissue of the front of the thorax seems a plexus of veins and the epigastric vessels are as large as the index-finger. Such instances are, I think, more common in lymphadenoma than in sarcoma or cancer. In these cases of chronic obstruction

* Fothergillian Prize Essay of the Medical Society of London, Philadelphia, 1889.

the finger-tips may be clubbed. There may be bulging of the sternum or the tumor may erode the bone and form a prominent subcutaneous growth. The rapidly growing lymphoid tumors more commonly than others perforate the chest wall. In four of thirteen cases of Hodgkin's disease, of which I have notes, there was mediastinal growth, and in three instances the sternum was eroded and perforated. The perforation may be on one side of the breast-bone. The projecting tumor may pulsate like an aneurism; the heart may be dislocated and its impulse much out of place. Contraction of one side of the thorax has been noted in a few instances. On palpation the fremitus is absent wherever the tumor reaches the chest wall. If pulsating, it rarely has the forcible, heaving impulse of an aneurismal sac. On auscultation there is usually silence over the dull region. The heart-sounds are not transmitted and the respiratory murmur is feeble or inaudible, rarely bronchial. Vocal resonance is, as a rule, absent. Signs of pleural effusion occur in a great many instances of mediastinal growth, and if in any doubt the aspirator needle should be used.

The *diagnosis* of mediastinal tumor from aneurism is sometimes extremely difficult. An interesting case reported and figured by Sokolosski, in Bd. 19 of the Deutsches Archiv für klinische Medicin, in which Oppolzer diagnosed aneurism and Skoda mediastinal tumor, illustrates how in some instances the most skilful of observers may be unable to agree. Scarcely a sign is found in aneurism which may not be duplicated in mediastinal tumor. This is not strange, since the symptoms in both are largely due to pressure. The time element is important. If a case has persisted for more than eighteen months the disease is probably aneurism. There are, however, exceptions to this. In the case of compression of the vena cava mentioned above, the disease has lasted for more than two years and the patient has improved so markedly under the use of arsenic that had he no other lymphatic enlargements the diagnosis might be uncertain. By far the most valuable sign of aneurism is the diastolic shock so often to be felt, and in a majority of cases to be heard, over the sac. This is rarely, if ever, present in mediastinal growths, even when they perforate the sternum and have communicated pulsation. Another point of importance is that in a tumor, advancing from the mediastinum, eroding the sternum and appearing externally, if aneurismal, has forcible, heaving, and distinctly expansile pulsations. The radiating pain in the back and arms and neck is rather in favor of aneurism, as is also a beneficial influence on it of iodide of potassium.

The frequency of pleural effusion in connection with mediastinal tumor is to be constantly borne in mind. It may give curiously complex characters to the physical signs—characters which are profoundly modified after aspiration of the liquid.

(4) **Abscess of the Mediastinum.**—Hare collected 115 cases of mediastinal abscess, in 77 of which there were details sufficient to permit the analysis. Of these cases the great majority occurred in males. Forty-four

were instances of acute abscess. The anterior mediastinum is most commonly the seat of the suppuration. The cases are most frequently associated with trauma. Some have followed erysipelas or occurred in association with eruptive fevers. Many cases, particularly the chronic abscesses, are of tuberculous origin. Of *symptoms*, pain behind the sternum is the most common. It may be of a throbbing character, and in the acute cases is associated with fever, sometimes with chills and sweats. If the abscess is large there may be dyspnœa. The pus may burrow into the abdomen, perforate through an intercostal space, or it may erode the sternum. Instances are on record in which the abscess has discharged into the trachea or œsophagus. In many cases, particularly of chronic abscess, the pus becomes inspissated and produces no ill effect. The *physical signs* may be very indefinite. A pulsating and fluctuating tumor may appear at the border of the sternum or at the sternal notch. The absence of *bruit*, of the diastolic shock, and of the expansile pulsation usually enables a correct diagnosis to be made. When in doubt a fine hypodermic needle may be inserted.

(5) **Miscellaneous Affections.**—In Hare's monograph there were 7 instances of fibroma, 11 cases of dermoid cysts, 8 cases of hydatid cysts, and cases of lipoma and gummata.

The *thymus gland* may be enlarged and produce the physical signs of mediastinal tumor. In children there are instances of spasm of the glottis, which is believed by some to depend upon enlargement of the thymus. Jacobi,* in his monograph, says that some instances of sudden death and also so-called thymic asthma may occasionally be referred to this cause. Malignant tumors of the thymus may attain considerable size and produce signs of tumor. In rare cases mediastinal growths develop from the *thyroid gland.* These may be substernal in position and directly connected with the gland. Kretschy has reported a sarcoma of the thyroid four and three quarter inches in length, which forms a mediastinal tumor passing to the level of the ninth dorsal vertebra. I have reported a somewhat similar instance, which developed in the left lobe of the thyroid and formed an elongated mass which passed down beside the trachea to the bifurcation.

(6) **Emphysema of the Mediastinum.**—Air in the cellular tissues of the mediastinum is met with in cases of trauma and occasionally in fatal cases of diphtheria and in whooping-cough. Champneys has called attention to its frequency in tracheotomy, in which he says the conditions favoring the production are division of the deep fascia, obstruction to the air-passages, and inspiratory efforts. The deep fascia, he says, should not be raised from the trachea. It is often associated with pneumothorax. The condition seems by no means uncommon. Angel Money found it in 16 of 28 cases of tracheotomy, and in two of these pneumothorax also was present.

* Transactions of the Association of American Physicians, vol. iii.

SECTION V.

DISEASES OF THE CIRCULATORY SYSTEM.

I. DISEASES OF THE PERICARDIUM.

I. PERICARDITIS.

PERICARDITIS is the result of infective processes, primary or secondary, or arises by extension of inflammation from contiguous organs.

Etiology.—*Primary*, so-called idiopathic, inflammation of this membrane is rare; but cases are met with, most commonly in children, in which there is no evidence of rheumatism or other conditions with which the disease is usually associated.

Pericarditis from injury usually comes under the care of the surgeon in connection with the primary wound. Interesting cases are those in which the traumatism is from within, due to the passage of some foreign body—such as a needle, a pin, or a bone—through the œsophagus into the pericardium.

As a *secondary* process pericarditis is met with in the following affections: (*a*) A majority of the cases occur in connection with rheumatism. The percentage given by different authors ranges from thirty to seventy. The articular trouble may be slight or, indeed, the disease may be associated with acute tonsillitis of rheumatic subjects. Cases are recorded in which the pericarditis has preceded the articular disease. (*b*) Septic processes rank next to rheumatism. In the acute necrosis of bone and puerperal fever it is not uncommon. (*c*) Tuberculosis, in which the disease may be primary or part of a general involvement of the serous sacs or associated with extensive pulmonary disease. (*d*) Eruptive fevers. In children, the disease is not infrequent after scarlatina. It is rarely met with in measles, small-pox, or typhoid fever. In other infective diseases, such as diphtheria and pneumonia, it is rare. (*e*) Dyscrasias. Certain altered conditions of the system seem to render the pericardium more susceptible to inflammation. Of these gout takes the first place. In chronic Bright's disease pericarditis is by no means rare. The *pericardite brightique* of the French forms one of the most important groups of the disease in persons over fifty years of age, most frequently accom-

panying the chronic interstitial form. Pericarditis has been met with also in scurvy and diabetes.

Pericarditis by extension of disease from contiguous organs. In pleuro-pneumonia it forms one of the most serious complications, and was present in 5 cases in 100 post-mortems in this disease which I made at the Montreal General Hospital. It is most often met with in the pleuro-pneumonia of children and of alcoholics. The association with simple pleurisy is much less common. In ulcerative endocarditis, purulent myocarditis, and in aneurism of the aorta pericarditis is occasionally found. It may also result from extension of disease from the bronchial glands, the ribs, sternum, vertebræ, and even from the abdominal viscera.

Pericarditis occurs at all ages. Cases are reported in the fœtus. In the new-born it may result from septic infection through the navel. Throughout childhood the incidence of rheumatism and scarlet fever makes it a frequent affection, whereas late in life it is most often associated with Bright's disease and gout. Males are somewhat more frequently attacked than females. Climatic and seasonal influences have been mentioned by some writers. The so-called epidemics of pericarditis have been outbreaks of pneumonia with this as a frequent complication.

Anatomically as well as clinically the disease may be considered under the following divisions:

1. Acute, plastic, or dry pericarditis.
2. Pericarditis with effusion—sero-fibrinous, hæmorrhagic, or purulent.
3. Chronic adhesive pericarditis (adherent pericardium).

Acute Plastic Pericarditis.—This, the most common form, occurs usually as a secondary process, and is distinguished by the small amount of fluid exudation, which does not, as in the next variety, give special characters to the disease. It is a benign form and rarely, if ever, of itself proves fatal.

Anatomically it may be partial or general. In the mildest grades the serous membrane looks lustreless and roughened. This is due to the presence of a thin fibrinous sheeting, which can be lifted with the knife, showing the membrane beneath to be injected or in places ecchymotic. As the fibrinous sheeting increases in thickness the constant movement of the adjacent surfaces gives to it sometimes a ridge-like, at others a honeycombed appearance. With more abundant fibrinous exudation the membranes present an appearance resembling buttered surfaces which have been drawn apart. The fibrin is in long shreds, and the heart presents a curiously shaggy appearance—the so-called hairy heart of old writers—*cor villosum*.

In mild grades the subjacent muscle looks normal; but in the more prolonged and severe cases there is myocarditis, and for 2 or 3 mm. beneath the visceral layer the muscle presents a pale, turbid appearance.

Many of these acute cases are tuberculous; covered by the layers of lymph the granulations are easily overlooked in a superficial examination.

Slight fluid exudation is invariably present, entangled in the meshes of fibrin, but there may be very thick fibrinous layers without much serous effusion.

Symptoms.—The majority of cases of simple plastic pericarditis, like simple endocarditis, present no symptoms, and unless sought for there are no objective signs indicating its existence. In the post-mortem room it is not uncommon to find it in cases in which its presence has been unsuspected during life.

Pain is a variable symptom, not usually intense, and in this form rarely excited by pressure. It is more marked in the early stage, and may be referred either to the præcordia or to the region of the xiphoid cartilage. Instances are recorded of pain of an aggravated and most distressing character resembling angina. Fever is usually present, but it is not always easy to say how much depends upon the primary febrile affection, and how much upon the pericarditis. It is as a rule not high, rarely exceeding 102·5°. In rheumatic cases hyperpyrexia has been observed.

Physical Signs.—*Inspection* is negative; *palpation* may reveal the presence of a distinct fremitus caused by the rubbing of the roughened pericardial surfaces. This is usually best marked over the right ventricle. It is not always to be felt, even when the friction sound on auscultation is loud and clear. *Auscultation:* The friction sound, due to the movement of the pericardial surfaces upon each other, is one of the most distinctive of physical signs. It is double, corresponding to the systole and diastole; but the synchronism with the heart-sounds is not accurate, and the to-and-fro murmur usually outlasts the time occupied by the first and second sound. In rare instances the friction is single; more frequently it appears to be triple in character—a sort of canter rhythm. The sounds have a peculiar rubbing, grating quality, characteristic when once recognized, and rarely simulated by endocardial murmurs. Sometimes instead of grating there is a creaking quality—the *bruit de cuir neuf*—the new-leather murmur of the French. The pericardial friction appears superficial, very close to the ear, and is usually intensified by pressure with the stethoscope. It is best heard over the right ventricle, the part of the heart which is most closely in contact with the front of the chest—that is, in the fourth and fifth interspaces and adjacent portions of the sternum. There are instances in which the friction is most marked at the base over the aorta and at the superior reflection of the pericardium. Occasionally it is best heard at the apex. It may be limited and heard over a very narrow area, or it may be transmitted up and down the sternum. There are, however, no definite lines of transmission as in the endocardial murmur. An important point is the variability of sounds, both in position and quality; they may be heard at one visit and not at another. The maximum of intensity will be found to vary with position.

Diagnosis.—There is rarely any difficulty in determining the presence of a dry pericarditis, for the friction sounds are distinctive. The double murmur of aortic incompetency may simulate closely the to-and-fro pericardial rub. I recall one instance at least in which this mistake was made. The constant character of the aortic murmur, the direction of transmission, the phenomena in the arteries, and the associated conditions of the disease should be sufficient to prevent this error.

I have never known an instance in which pericarditis was mistaken for endocarditis, though writers refer to such, and give the differential diagnosis in the two affections. The only possible mistake could be made in those rare instances of single soft, systolic, pericardial friction.

Pleuro-pericardial friction is very common, and may be associated with endo-pericarditis, particularly in cases of pleuro-pneumonia. It is frequent, too, in phthisis. It is best heard over the left border of the heart, and is much affected by the respiratory movement. Holding the breath or taking a deep inspiration may annihilate it. The rhythm is not the simple to-and-fro diastolic and systolic, but the respiratory rhythm is superadded, usually intensifying the murmur during expiration and lessening it on inspiration. In phthisis there are instances in which, with the friction, a loud systolic click is heard, due to the compression of a thin layer of lung and the expulsion of a bubble of air from a small softening focus or from a bronchus.

Course and Termination.—Simple fibrinous pericarditis never kills, but it occurs so often in connection with serious affections that we have frequent opportunities to see all stages of its progress. In the majority of cases the inflammation subsides and the thin fibrinous laminæ gradually become converted into connective tissue, which unites the pericardial leaves firmly together. In other instances the inflammation progresses, with increase of the exudation, and the condition is changed from a "dry" to a "moist" pericarditis, or the pericarditis with effusion.

In a few instances—probably always tuberculous—the simple plastic pericarditis becomes chronic, and great thickening of both visceral and parietal layers is gradually induced.

Pericarditis with Effusion.—Though commonly a direct sequence of the dry or plastic pericarditis, of which it is sometimes spoken as the second stage, this form presents special features and deserves separate consideration. It is found most frequently in association with acute rheumatism, tuberculosis, and septicæmia, and sets in usually with the symptoms above described, namely, præcordial pain, with slight fever or a distinct chill.

In children the disease may, like pleurisy, come on without local symptoms, and, after a week or two of failing health, slight fever, shortness of breath, and increasing pallor, the physician may find, to his astonishment, signs of most extensive pericardial effusion. These latent cases are often tu-

berculous. The effusion may be sero-fibrinous, hæmorrhagic, or purulent. The amount varies from 200 or 300 c. c. to 2 litres. In the cases of sero-fibrinous exudation the pericardial membranes are covered with thick, creamy fibrin, which may be in ridges or honeycombed, or may present long, villous extensions. The parietal layer may be several millimetres in thickness and may form a firm, leathery membrane. The hæmorrhagic exudation is usually associated with tuberculous, or with cancerous pericarditis, or with the disease in the aged. The lymph is less abundant, but both surfaces are injected and often show numerous hæmorrhages. Thick, curdy masses of lymph are usually found in the dependent part of the sac. In the purulent effusion the fluid has a creamy consistency, particularly in tuberculosis. In many cases the effusion is really sero-purulent, a thin, turbid exudation containing flocculi of fibrin.

The pericardial layers are greatly thickened and covered with fibrin. When the fluid is pus, they present a grayish, rough, granular surface. Sometimes there are distinct erosions on the visceral membrane. The heart muscle in these cases becomes involved to a greater or less extent, and on section, the tissue, for a distance of from two to three millimetres, is pale and turbid, and shows evidence of fatty and granular change. Endocarditis coexists frequently, but rarely results from the extension of the inflammation through the wall of the heart.

Symptoms.—Even with copious effusion the onset and course may be so insidious that no suspicion of the true nature of the disease is aroused.

As in the simple pericarditis, pain may be present, either sharp and stabbing or as a sense of distress and discomfort in the cardiac region. It is more frequent with effusion than in the plastic form. Pressure at the lower end of the sternum usually aggravates it. Dyspnœa is a common and important symptom, one which, perhaps, more than any other, excites suspicion of grave disorder and leads to careful examination of heart and lungs. The patient is restless, lies upon the left side or, as the effusion increases, sits up in bed. Associated with the dyspnœa is in many cases a peculiarly dusky, anxious countenance. The pulse is rapid, small, sometimes regular, and may present the characters known as *pulsus paradoxus*, in which during each inspiration the pulse-beat becomes very weak or is lost. These symptoms are due, in great part, to the direct mechanical effect of the fluid within the pericardium which embarrasses the heart's action. Other pressure effects are distention of the veins of the neck, dysphagia, which may be a marked symptom, and irritative cough from compression of the trachea. Aphonia is not uncommon, due to compression or irritation of the recurrent laryngeal as it winds round the aorta. Another important pressure effect is exercised upon the left lung. In massive effusion the pericardial sac occupies such a large portion of the antero-lateral region of the left side that the condition has frequently been mistaken for pleurisy. Even in moderate grades the left

lung is somewhat compressed. This is an additional element in the production of the dyspnœa.

Great restlessness, insomnia, and in the later stages low delirium and coma are symptoms in the more severe cases. Delirium and marked cerebral symptoms are associated with the hyperpyrexia of rheumatic cases, but apart from the ordinary delirium there may be peculiar mental symptoms. The patient may become melancholic and show suicidal tendencies. In other cases the condition resembles closely delirium tremens. Sibson, who has specially described this condition, states that the majority of such cases recover. Chorea may also occur, as was pointed out by Bright. Epilepsy is a rare complication which has occurred, as in pleurisy, during paracentesis.

Physical Signs.—*Inspection.*—In children the præcordia bulges and with copious exudation the antero-lateral region of the left chest becomes enlarged. The intercostal spaces are prominent and there may be marked œdema of the wall. Perforation externally through a space is very rare. Owing to the compression of the lung, the expansion of the left side is greatly diminished. The diaphragm and left lobe of the liver may be pushed down and may produce a distinct prominence in the epigastric region.

Palpation.—A gradual diminution and final obliteration of the cardiac shock is a striking feature in progressive effusion. The apex beat is often raised an interspace and dislocated outward. Alteration in the position of the impulse simultaneously with the position of the patient, a sign upon which Oppolzer laid great stress, cannot often be determined, as the beat may, and usually does, disappear entirely. The pericardial friction may lessen with the effusion, though it often persists at the base when no longer palpable over the right ventricle, or may be felt in the erect and not in the recumbent posture. Fluctuation can rarely, if ever, be detected.

Percussion gives most important indications. The gradual distention of the pericardial sac pushes aside the margins of the lungs so that a large area comes in contact with the chest wall and gives a greatly increased percussion dulness. The form of this dulness is irregularly pear-shaped; the base or broad surface directed downward and the stem or apex directed upward toward the manubrium.

Auscultation.—The friction sound heard in the early stages may disappear when the effusion is copious, but often persists at the base or at the limited area of the apex. It may be audible in the erect and not in the recumbent posture. With the absorption of the fluid the friction returns. One of the most important signs is the gradual weakening of the heart-sounds, which with the increase in the effusion may become so muffled and indistinct as to be scarcely audible. The heart's action is usually increased and the rhythm disturbed. Occasionally a systolic endocardial murmur is heard.

Important accessory signs in large effusion are due to pressure on the left lung. The antero-lateral margin of the lower lobe is pushed aside and in some instances compressed, so that percussion in the axillary region, in and just below the transverse nipple line, gives a modified percussion note, usually a flat tympany. Variations in the position of the patient may change materially this modified percussion area, over which on auscultation there is either feeble or tubular breathing.

Course.—Cases vary extremely in the rapidity with which the effusion takes place. In every instance, when a pericardial friction murmur has been detected, the practitioner should immediately outline with care—using the aniline pencil or nitrate of silver—the upper and left limits of cardiac dulness, since he will in this way have certain positive guides in determining the rate and grade of the effusion. In many instances the exudation is slight in amount, reaches a maximum within forty-eight hours, and then gradually subsides. In other instances the accumulation is more gradual and progressive, increasing for several weeks. To such cases the term chronic has been applied. The rapidity with which a sero-fibrinous effusion may be absorbed is surprising. The possibility of the absorption of purulent exudate is shown by the cases in which the pericardium contains semi-solid grayish masses in all stages of calcification. With sero-fibrinous effusion, if moderate in amount, recovery is the rule, with inevitable union, however, of the pericardial layers. In some of the septic cases there is a rapid formation of pus and a fatal result may follow in three or four days. More commonly, when death occurs with large effusion, it is not until the second or third week and takes place by gradual asthenia.

Prognosis.—In the sero-fibrinous effusions the outlook is good, and a large majority of all the rheumatic cases recover. The purulent effusions are, of course, more dangerous; the septic cases are usually fatal, and recovery is rare in the slow, insidious tuberculous forms.

Diagnosis.—Probably no serious disease is so frequently overlooked by the practitioner. Post-mortem experience shows how often pericarditis is not recognized, or goes on to resolution and adhesion without attracting notice. In a case of rheumatism, watched from the outset, with the attention directed daily to the heart, it is one of the simplest of diseases to diagnose, but when one is called to a case for the first time and finds perhaps an increased area of precordial dulness, it is often very hard to determine with certainty whether or not effusion is present.

The difficulty usually lies in distinguishing between dilatation of the heart and pericardial effusion. Although the differential signs are simple enough on paper, it is notoriously difficult in certain cases, particularly in stout persons, to say which of the conditions exists. The points which deserve attention are:

(*a*) The character of impulse, which in dilatation, particularly in thin-chested people, is commonly visible and wavy.

(*b*) The shock of the cardiac sounds is more distinctly palpable in dilatation.

(*c*) The area of dulness in dilatation rarely has the triangular form; nor does it, except in cases of mitral stenosis, reach so high along the left sternal margin or so low in the fifth and sixth interspaces *without visible or palpable impulse.* An upper limit of dulness shifting with the position speaks strongly for effusion.

(*d*) In dilatation the heart-sounds are clearer, often sharp, valvular, or fœtal in character; whereas in effusion the sounds are distant and muffled.

(*e*) Rarely in dilatation is the distention sufficient to compress the lung and produce the tympanitic note in the axillary region.

The number of excellent observers who have acknowledged that they have failed sometimes to discriminate between these two conditions, and who have indeed performed paracentesis *cordis* instead of paracentesis *pericardii*, is perhaps the best comment on the difficulties which certain cases present.

Massive (1½ to 2 l.. xudations have been confounded with a pleural effusion. On more than one occasion the pericardium has been tapped under the impression that the exudate was pleuritic. The flat tympany in the infrascapular region, the absence of well-defined movable dulness, and the feeble, muffled sounds are indicative points. If the case has been followed from day to day there is rarely much difficulty; but it is different when a case presents a large area of dulness in the antero-lateral region of the left chest, and there is no to-and-fro pericardial friction murmur. Many of the cases have been regarded as encapsulated pleural effusion.

The nature of the fluid cannot positively be determined without aspiration; but a fairly accurate opinion can be formed by the nature of the primary disease and the general condition of the patient. In rheumatic cases the exudation is usually sero-fibrinous; in septic and tuberculous cases it is often purulent from the outset; in senile, nephritic, and tuberculous cases the exudation is sometimes hæmorrhagic.

Treatment.—The patient should have absolute quiet, mentally and bodily, so as to reduce to a minimum the heart's action. Drugs given for this purpose, such as aconite or digitalis, are of doubtful utility. Local bloodletting by cupping or leeches is certainly advantageous in robust subjects, particularly in the cases of extension in pleuro-pneumonia. The ice-bag or Leiter's tube may be used to advantage. They have the double effect of reducing the heart's action and retarding the progress of inflammation. Blisters are not indicated in the early stage.

When effusion is present, the following measures to promote absorption may be adopted: Blisters to the præcordia, a practice not so much in vogue now as formerly. It is surprising, however, in some instances, how quickly an effusion will subside on their application. If the patient's

strength is good, a purge every other morning may be given. The diet should be light, dry, and nutritious. In cases in which the pulse is strong and the constitutional disturbance not great, iodide of potassium may be of service, and the action of the kidneys may be promoted by the infusion of digitalis and acetate of potash.

When the effusion is large, as soon as signs of serious impairment of the heart occur, as indicated by dyspnœa, small rapid pulse, dusky, anxious countenance, surgical measures should be resorted to, and paracentesis, or incision of the pericardium, at once be performed. With the sero-fibrinous exudate, such as commonly occurs after rheumatism, aspiration is sufficient; but when the exudate is purulent the pericardium should be freely incised and freely drained. The puncture may be made in the fourth interspace, either at the left sternal margin or 2·5 cm. (an inch) from it. If made in the fifth interspace it is well to puncture an inch and a half from the left sternal margin. In large effusions the pericardium can also be readily reached without danger by thrusting the needle upward and backward close to the costal margin in the left costo-xiphoid angle. The results of paracentesis of the pericardium have so far not been satisfactory. With an earlier operation in many instances and a more radical one in others—a free incision and not aspiration when the fluid is purulent—the percentage of recoveries will be greatly increased.

Chronic Adhesive Pericarditis (*Adherent Pericardium*).—This condition follows acute pericarditis, and may be partial or universal. It is not very uncommon to meet with limited synechia over the right ventricle. In the mildest grades of complete adhesion the amount of connective tissue between the membranes is slight, and there is not much thickening. These are the instances which follow the fibrinous rheumatic pericarditis. The most extreme thickening of the membranes is met with in the chronic tuberculous form, which has already been described, and which is much more common than indicated in the literature. After the absorption of an extensive purulent or sero-purulent exudate the inspissated remnants may undergo calcification. This may be in quite a limited region, most frequently over the auricles or at the base of the heart. In extreme grades the organ is completely invested by a calcareous membrane, which in places may be from 1 to 1·5 cm. in thickness.

The *symptoms* of adherent pericardium are uncertain and indefinite. A majority of the cases are met with accidentally in the post-mortem room, and there may have been no indications whatever during life of cardiac disturbance. Enlargement of the heart is an almost constant accompaniment of universal adhesion, and many of the cases come under observation for the first time with failure of this hypertrophy and signs of cardiac insufficiency.

The following are the important points in the diagnosis:

(1) *Inspection.*—In children, in whom the condition is not very un-

common as a sequence of rheumatism, the hypertrophied heart causes bulging of the chest wall. The area of cardiac impulse is increased and may sometimes be seen from the third to the sixth interspace and beyond the nipple line. The strongest impulse may be to the right of the apex. The wavy character of the pulsation in the third, fourth, and fifth interspaces is not peculiar to adherent pericardium. Not much stress can be laid upon the fixed position of the impulse, which in great enlargement of the heart is not much influenced either by posture or respiration. A more important point is systolic retraction of the apex region. Whether this occurs without adhesion of the pericardium to the chest wall is doubtful. It is often marked, and is sometimes best appreciated by the application of the hand over the apex region, which is felt to be drawn in at the moment of systole. The retraction may be most noticeable in the lower sternal region or even at the xiphoid cartilage. Following this there is sometimes a rapid rebound—the diastolic shock—which has been regarded by some as the most reliable of all signs of pericardial adhesion. Associated with this diastolic rebound is the so-called *Friedrich's sign*—diastolic collapse of the cervical veins.

(2) *Percussion* reveals an increase in the area of cardiac dulness, particularly upward as high as the second interspace. In a majority of the cases there are adhesions as well between the pleura and pericardium—in ten of thirteen cases analyzed by Ord. In some instances the dulness may reach as high as the first interspace. A sign of value is the fixed limit above and to the left of cardiac dulness, as pointed out by C. J. B. Williams. When the outer layer of the pericardium is adherent to the pleura this is a sign of very definite value, and the limit of dulness varies very slightly on deep inspiration.

(3) On *auscultation* the phenomena vary extremely with the condition of the chambers. There may be no murmurs. When extreme dilatation is present the gallop or fœtal rhythm occurs. A loud regurgitant murmur is not uncommon at the apex region, and the cases are frequently mistaken for mitral insufficiency.

(4) The *pulsus paradoxus* in which during inspiration the pulse-wave is small and feeble, is sometimes present, but it is not a diagnostic sign of either simple pericardial adhesion or of the cicatricial mediastino-pericarditis.

Adherent pericardium with extreme dilatation of the heart may raise the suspicion of pericarditis with effusion, as the outline of dulness in both is somewhat alike. As a rule, however, the basic dulness is broader in adhesion, and has not the pear-shaped outline. The extent and wavy character of the impulse is never so marked in large effusions, and the heart-sounds are muffled.

II. OTHER AFFECTIONS OF THE PERICARDIUM.

1. **Hydropericardium.**—Naturally there are in the pericardial sac a few cubic centimetres of clear, citron-colored fluid, which probably represents a post-mortem transudate. In certain conditions during life there may be large secretions of serum forming what is known as dropsy of the pericardium. It occurs usually in connection with general dropsy, due to kidney or heart disease; more commonly the former. It rarely of itself proves fatal, though when the effusion is excessive it adds to the embarrassment of the heart and the lungs, particularly when the pleural cavities are the seat of similar exudation. There are rare instances in which effusion into the pericardium occurs after scarlet fever with few, if any, other dropsical symptoms. The physical signs are those already referred to in connection with pericarditis with effusion. It is frequently overlooked.

In rare cases the serum has a milky character—chylo-pericardium.

2. **Hæmo-pericardium.**—This condition, by no means uncommon, is met with in aneurism of the first part of the aorta, of the cardiac wall, or of the coronary arteries, and in rupture and wounds of the heart. Death usually follows before there is time for the production of symptoms other than those of rapid heart-failure due to compression. Particularly is this the case in aneurism. In rupture of the heart the patient may live for many hours or even days with symptoms of progressive heart-failure, dyspnœa, and the physical signs of effusion.

As already mentioned, the inflammatory exudate of tubercle or cancer is often blood-stained. The same is true of the effusion in the pericarditis of Bright's disease and of old people.

3. **Pneumo-pericardium.**—Gas is rarely found in the pericardial sac, and is due, as a rule, to perforation from without, as in the case of stab wounds, or the result of perforation from the lungs, œsophagus, or stomach. Possibly, too, it may result from the decomposition of a purulent exudate. As a result of perforation, acute pericarditis is always excited, and the effusion rapidly becomes purulent. The physical signs are remarkable. When the effusion is copious the fluid and gas together give a movable area of percussion dulness with marked tympany in the region of the gas. On auscultation, remarkable splashing, churning, metallic phenomena are heard with friction and possibly feeble, distant heart-sounds. Death follows rapidly, even in thirty-six hours, as in a case (the only one which I have seen) of perforation of the pericardium in cancer of the stomach. Except as a result of injury, the condition is not one for which treatment is available. In a case of perforation from without with signs of effusion, to enlarge the wound by free incision would be justifiable.

II. DISEASES OF THE HEART.

I. ENDOCARDITIS.

Inflammation of the lining membrane of the heart is usually confined to the valves, so that the term is practically synonymous with valvular endocarditis. It occurs in two forms—*acute*, characterized by the presence of vegetations with loss of continuity or of substance in the valve tissues; *chronic*, a slow sclerotic change, resulting in thickening, puckering, and deformity.

Acute Endocarditis.

This occurs in rare instances as a primary, independent affection; but in the great majority of cases it is an accident in various infective processes, so that in reality the disease does not constitute an etiological entity.

For convenience of description we speak of a simple or benign, and a malignant or ulcerative endocarditis, between which, however, there is no essential anatomical difference, as all gradations can be traced, and they represent but different degrees of intensity of the same process.

Simple Endocarditis.—This is characterized by the presence on the valves or on the lining membrane of the chambers of minute vegetations, ranging from 1 to 4 mm. in size, with an irregular and fissured surface, giving to them a warty or verrucose appearance. Often these little cauliflower-like excrescences are attached by very narrow pedicles. It is rare to see any swelling or infiltration of the endocardium in the neighborhood of even the smallest of the granulations, and although small capillary vessels do exist at the edges of the valves, redness, indicative of the injection or distention of the vessels, is extremely rare. With time the vegetations may increase greatly in size, but in what may be called simple endocarditis the size rarely exceeds that mentioned above. The finer changes in the process consist of the proliferation of the subendothelial connective-tissue elements, resulting in a small-celled infiltration. What part, if any, the endothelial cells play in this is not accurately known. The superficial elements undergo a coagulation necrosis, and fibrin is deposited from the blood, often in layers. Practically a vegetation is a small area of granulation tissue capped with fibrin. Micro-organisms are present, entangled in the granular and fibrillated fibrin, but whether they constitute an essential and constant element in all cases of simple endocarditis has not yet been decided.

The further changes in the vegetation may be either in the direction of increased proliferation of the connective-tissue elements of the valve, forming an extensive area of necrosis and the production of the condition which, from its more intense grade, we speak of as malignant or ulcerative endocarditis; or, as is more usual, healing occurs. The vegetation is

absorbed, and there remains a small nodular thickening of the valve. A third possibility is the dislocation of a vegetation with transference as an embolus to a distant part of the circulation. It is to be noted, however, that this untoward event is rare in acute endocarditis associated with febrile affections, whereas it is by no means uncommon in the simple endocarditis which occurs so constantly on old sclerotic valves.

Anatomically, in the majority of instances of acute endocarditis, cicatrization of the granulation tissue takes place in time, with but little damage to the valve beyond slight nodular thickening. The essential danger is remote and results from the slow changes in the valve tissue, which are so apt to follow an acute inflammation. Why this should be so cannot at present be explained; but the fact remains that the simple endocarditis, harmless in itself, such as we meet with in rheumatism or in chorea, lays the foundation of subsequent organic lesions, owing to the initiation of nutritive changes leading to sclerosis with contraction and deformity.

Endocarditis is much more common on the left side of the heart and involves the valvular endocardium in the great majority of cases. During fœtal life the right side of the heart is often affected. The chordæ tendineæ are sometimes involved with the valves, rarely alone. The mitral valves are more often affected than the aortic. On the mitral segment the vegetations are usually on the auricular face, not at the margin, but at a distance of 2 or 3 mm., forming a row of bead-like outgrowths. So, too, on the aortic segment they are not seen on the free margin, but just below, on the ventricular face, following the margin of the so-called lunated spaces. In both the valves this peculiar distribution follows, as Sibson suggests, the lines of maximum contact.

Etiology.—Simple endocarditis does not constitute a disease of itself, but is invariably found with some other affection. The general experience of the profession has confirmed the original observation of Bouillaud as to the frequency of association of simple endocarditis with acute articular rheumatism. Possibly it is nothing in the disease itself, but simply an altered state of the fluid media—a reduction perhaps of the lethal influences which they normally exert—permitting the invasion of the blood by certain micro-organisms. Tonsillitis, which in some forms is regarded as a rheumatic affection, may be complicated with endocarditis. Of the specific diseases of childhood it is not uncommon in scarlet fever, while it is rare in measles and chicken-pox. In diphtheria simple endocarditis is rare. It was not present in a single instance of 30 autopsies which I made in this disease at the Montreal General Hospital. In small-pox it is not common. It is stated to be more frequent in typhoid fever but was not present in 65 post-mortems in this disease.

In pneumonia both simple and malignant endocarditis are common. In 100 autopsies in this disease made at the Montreal General Hospital there were 5 instances of the former. Acute endocarditis is by no

means rare in phthisis. I have met with it in 12 cases in 216 post-mortems.

In chorea simple warty vegetations are found on the valves in a large majority of all fatal cases. There is no disease in which, post mortem, acute endocarditis has been so frequently found. And lastly, simple endocarditis is met with in diseases associated with loss of flesh and progressive debility, as cancer, and such disorders as gout, diabetes, and Bright's disease.

A very common form is that which occurs on the sclerotic valves in old heart-disease—the so-called recurring endocarditis.

Symptoms.—Neither the clinical course nor the physical signs are in any respect characteristic. The great majority of the cases are latent and there is no indication whatever of cardiac mischief. Experience has taught us that endocarditis is frequently found post mortem in persons in whom it was not suspected during life. There are certain features, however, by which its presence is indicated with a degree of probability. The patient, as a rule, does not complain of any pain or cardiac distress. In a case of acute rheumatism, for example, the symptoms to excite suspicion would be increased rapidity of the heart's action, perhaps slight irregularity, and an increase in the fever without aggravation of the joint trouble. Rows of tiny vegetations on the mitral or on the aortic segments seem a trifling matter to excite fever and it is difficult in the endocarditis of febrile processes to say definitely in every instance that an increase in the fever depends upon the endocardial complication. But a study of the recurring endocarditis—which is of the warty variety, consisting of minute beads on old sclerotic valves—shows that this process may be associated, for days or week at a time, with slight fever ranging from 100° to 102½°. Palpitation may be a marked feature and is a symptom upon which certain authors lay great stress.

The *diagnosis* of the condition rests upon physical signs which are notoriously uncertain. The presence of a murmur at one or other of the cardiac areas in a case of fever is often regarded as indicative of the existence of endocarditis. This extremely common mistake has arisen from the fact that the *bruit de souffle* or bellows murmur is common to endocarditis and a number of other conditions which have nothing to do with it. At first there may be only a slight roughening of the first sound, which may gradually develop into a distinct murmur. Taken alone, it is, however, a very uncertain and fallacious sign.

Malignant Endocarditis.—Acute endocarditis of a malignant character is met with:

(*a*) As a primary disease of the lining membrane of the heart or of its valves.

(*b*) As a secondary affection in acute rheumatism, pneumonia, and in various specific fevers; or as an associated condition in septic processes.

It is also known by the names of ulcerative, infectious, or diphtheritic endocarditis, but the term malignant seems most appropriate to characterize the essential clinical features of the disease.

Etiology.—The existence of a primary endocarditis has been doubted; but there are instances in which persons previously in good health, without any history of affections with which endocarditis is usually associated, have been attacked with symptoms resembling severe typhus or typhoid. In one case which I saw death occurred on the sixth day and no lesions were found other than those of malignant endocarditis.

Rheumatism, with which simple endocarditis is frequently associated, is not so often complicated with the malignant form. Thus, in only 24 of 209 cases the symptoms of severe endocarditis arose in the progress of acute or subacute rheumatism. In only 3 of the Montreal cases was there a history of rheumatism either before or during the attacks.

Malignant endocarditis is extremely rare in chorea. Of all acute diseases complicated with severe endocarditis pneumonia probably heads the list. This fact, which had been referred to by several of the older writers, was brought out in a striking manner by the figures on which my lectures were based. In 11 of the 23 Montreal cases the disease came on with lobar pneumonia, while it developed with this disease in 54 of the 209 cases analyzed—indeed, the endocarditis which occurs in pneumonia seems to be of an unusually malignant type, as in 16 cases of my 100 autopsies in this disease in which this lesion was present, 11 were of this form. Meningitis was associated with endocarditis in 25 of the 209 cases, and in 15 there was also pneumonia.

The affection may complicate erysipelas, septicæmia (from whatever cause), and puerperal fever and gonorrhœa. Malignant endocarditis is very rare in tuberculosis, typhoid fever, and diphtheria.

It has been stated by many writers that endocarditis occurs in ague. With the unusual facilities for the study of this disease which I have had in the past seven years I have not yet met with an instance. Unquestionably, in the majority of these cases, the intermittent pyrexia, which has been regarded as characteristic of the ague, has depended upon the endocarditis. In dysentery cases have been described. In small-pox and scarlet fever, with which simple endocarditis is not infrequently complicated, the malignant form is extremely rare.

Morbid Anatomy.—The lesions may be either vegetative, ulcerative, or suppurative, and these forms may occur alone or in combination. Even with vegetations there is distinct necrosis and loss of the endocardial substance. More frequently there is ulceration, either superficial, involving only the endocardium, or deep and distinct, leading to perforation of a valve, of a septum, or even of the heart itself. In the suppurative form the deeper tissues of the valve appear first affected and small abscesses are found at the bases of the vegetations. The vegetations may present a remarkable greenish-gray or greenish-yellow color, and when of long stand-

ing, or even in cases which from the clinical history appear to be tolerably acute, the vegetations may be crusted with lime salts.

A large vegetation of malignant endocarditis consists histologically of a granular and fibrillated fibrin, colonies of micro-organisms, and distinct granulation tissue at the base, while the subjacent endocardial layers show infiltration and proliferation. The destruction of tissue results from a gradual extension of the necrotic processes. Various micro-organisms have been found in connection with the disease, and the following brief statement may be made with reference to them: In a large proportion of the cases streptococci and staphylococci are found. The pneumococcus has been cultivated from the vegetations in pneumonia. Other forms have occasionally been met with.

The following figures, taken from my Gulstonian lectures at the Royal College of Physicians, give an approximate estimate of the frequency with which in 209 cases different parts of the heart were affected: Aortic and mitral valves together, 41; aortic valves alone, 53; mitral valves alone, 77; tricuspid in 19; the pulmonary valves in 15; and the heart wall in 33. In 9 instances the right heart alone was involved.

Mural endocarditis is seen most often at the upper part of the septum of the left ventricle. Next in order is the endocarditis of the left auricle on the postero-external wall. The ulcerative changes may lead to perforation of a valve segment, erosion of the chordæ tendineæ, perforation of the septum, or even of the heart itself. A common result of the ulceration is the production of valvular aneurism. In three fourths of the cases the affected valves present old sclerotic changes. The process may extend to the aorta, producing, as in one of my cases, extensive endarteritis with multiple acute aneurisms.

The associated pathological changes are partly those of the primary disease to which the endocarditis is secondary and partly those due to embolism. In the endocarditis of septic processes there is the local lesion —an acute necrosis, a suppurative wound, or puerperal disease. In many cases the lesions are those of pneumonia, rheumatism, or other febrile processes. The changes due to embolism constitute the most striking features, but it is remarkable that in some instances, even with endocarditis of a markedly ulcerative character, there may be no trace of embolic processes.

The infarcts may be few in number—only one or two, perhaps, in the spleen or kidney—or they may exist in hundreds throughout the various parts of the body. They may present the ordinary appearance of red or white infarcts of a suppurative character. They are most common in the spleen and kidneys, though they may be numerous in the brain, and in many cases are very abundant in the intestines. In right-sided endocarditis there may be infarcts in the lungs. In many of the cases there are innumerable miliary abscesses. Acute suppurative meningitis was met with in 5 of 23 of the Montreal cases, and in over ten per cent of the 209

cases analyzed in the literature. Acute suppurative parotitis also may occur.

Symptoms.—It is difficult to give a satisfactory clinical picture of the disease because the modes of onset are so varied and the symptoms so diverse. Arising in the course of some other disease, there may be simply an intensification of the fever or a change in its character. In a majority of the cases there are present certain general features, such as irregular pyrexia, delirium, sweating, gradual failure of strength.

Embolic processes may give special characters, such as delirium, coma or paralysis from involvement of the brain or its membranes, pain in the sides and local peritonitis from infarction of the spleen, bloody urine from implication of the kidneys, impaired vision from retinal hæmorrhage, and suppuration, and even gangrene, in various parts from the distribution of the emboli.

Two special types of the disease have been recognized—the septic or pyæmic and the typhoid. Other cases closely resemble true intermittent fever. In some the cardiac symptoms are most prominent, while in others again the main symptoms may be those of an acute affection of the cerebro-spinal system.

The *septic type* is met with usually in connection with an external wound, the puerperal process, or an acute necrosis. There are rigors, sweats, irregular fevers, and all of the signs of septic infection. The heart symptoms may be completely masked by the general condition, and attention called to them only on the occurrence of embolism. In a most remarkable sub-group of this type the disease may simulate a quotidian or a tertian ague. The symptoms may develop in persons with chronic heart-disease without any external lesions. These cases may be much prolonged —for three or four months, or even longer, as in a case of Bristowe's. The existence in some of these instances of a previous genuine malaria has been a very puzzling circumstance.

The *typhoid type* is by far the most common and is characterized by an irregular temperature, early prostration, delirium, somnolence, and coma, relaxed bowels, sweating, which may be of a most drenching character, petechial and other rashes, and occasionally parotitis. The heart symptoms may be completely overlooked, and in some intances the most careful examination has failed to discover a murmur.

Under the *cardiac group*, as suggested by Bramwell, may be considered those cases in which patients with chronic valve disease are attacked with marked fever and evidence of recent endocarditis. Many such cases present symptoms of the pyæmic and typhoid character and may run a most acute course. In others the process is less intense and the course more chronic, lasting for weeks or months, so that the term acute is scarcely applicable to them. In a case of this kind under the care of Mullin, of Hamilton, the irregular fever lasted for more than a year. The autopsy showed extensive vegetative and ulcerative disease of the mitral valves.

There are cases in which it is often difficult to decide whether malignant endocarditis is present or not. Thus, a patient with aortic valve disease is under treatment for failing compensation and begins to have irregular fever with restlessness and cardiac distress; embolic phenomena may develop—sudden hemiplegia, pain in the region of the spleen, or bloody urine, or perhaps peripheral embolism. There may be a low delirium and the case may run a tolerably acute course; but in other instances the fever subsides and recovery occurs.

In what may be termed the *cerebral group* of cases the clinical picture may simulate a meningitis, either basilar or cerebro-spinal. There may be acute delirium or, as in three of the Montreal cases, the patient may be brought into the hospital unconscious. Heineman reports an instance, with autopsy, in which the clinical picture was that of an acute cerebro-spinal meningitis.

Certain special symptoms may be mentioned. The fever is not always of a remittent type, but may be high and continuous. Petechial rashes are very common and render the similarity very strong to certain cases of typhoid and cerebro-spinal fevers. In one case the disease was thought to be hæmorrhagic small-pox. Erythematous rashes are not uncommon. The sweating may be most profuse, even exceeding that which occurs in phthisis and ague. Diarrhœa is not necessarily associated with embolic lesions in the intestines. Jaundice has been observed and cases are on record which were mistaken for acute yellow atrophy.

The heart symptoms may be entirely latent and are not found unless a careful search be made. Even on examination there may be no murmur present. Instances are recorded by careful observers, in which the examination of the heart has been negative. Cases with chronic valve disease usually present no difficulty in diagnosis.

The course of the disease is varied, depending largely upon the nature of the primary trouble. Except in the disease grafted upon chronic valvulitis the course is rarely extended beyond five or six weeks. As already mentioned, there are instances in which the disease is prolonged for months. The most rapidly fatal case on record is described by Eberth, the duration of which was scarcely two days.

Diagnosis.—In many cases the detection of the disease is very difficult; in others, with marked embolic symptoms, it is easy. From simple endocarditis it is readily distinguished, though confusion occasionally occurs in the transitional stage, when a simple is developing into a malignant form. The constitutional symptoms are of a graver type, the fever is higher, rigors are common, and septic and typhoid symptoms develop. Perhaps a majority of the cases not associated with puerperal processes or bone disease are confounded with typhoid fever. A differential diagnosis may even be impossible, particularly when we consider that in typhoid fever infarctions and parotitis may occur. The diarrhœa and abdominal tenderness may also be present, which with the stupor and progressive

asthenia make a picture not to be distinguished from this disease. Points which may guide us are: The more abrupt onset in endocarditis, the absence of any regularity of the pyrexia in the early stage of the disease, and the cardiac pain. Oppression and shortness of breath may be early symptoms in malignant endocarditis. Rigors, too, are not uncommon. Between pyæmia and malignant endocarditis there are practically no differential features, for the disease really constitutes an *arterial pyæmia* (Wilks). In the acute cases resembling malignant fevers, the diagnosis is usually made of typhus, typhoid, cerebro-spinal fever, or even of hæmorrhagic small-pox. The intermittent pyrexia, occurring for weeks or months, has led in some cases to the diagnosis of malaria, but this disease could now be positively excluded by the blood examination.

The cases usually terminate fatally. The instances of recovery are those more subacute forms, the so-called recurring endocarditis developing on old sclerotic valves in cases of chronic heart-disease.

Treatment.—We know no measures by which in rheumatism, chorea, or the eruptive fevers the onset of endocarditis can be prevented. As it is probable that many cases develop, particularly in children, in mild forms of these diseases, it is well to guard the patients against taking cold and insist upon rest and quiet, and to bear in mind that of all complications an acute endocarditis, though in its immediate effects harmless, is perhaps the most serious. This statement is enforced by the observations of Sibson that on a system of absolute rest the proportion of cases of rheumatism attacked by endocarditis was less than of those who were not so treated.

It is doubtful whether the salicylates in rheumatism have an influence in reducing the liability to endocarditis. When the endocarditis is present we know no remedies which will definitely influence the valvular lesions. If there is much vascular excitement aconite may be given and an ice-bag placed over the heart.

The salicylates are strongly advised by some writers and the sulphocarbolates have been recommended by Sansom. In the severer cases of malignant endocarditis the treatment is practically that of septicæmia.

Chronic Endocarditis.

This condition, which is a sclerosis of the valve, may be primary, but is oftener secondary to acute endocarditis, particularly the rheumatic form. It is essentially a slow, insidious process which leads to deformity of the valve segment and is the foundation of chronic valvular disease.

Certain poisons appear capable of initiating the change, such as alcohol, syphilis, and gout, though we are at present ignorant of the way in which they act. A very important factor, particularly in the case of the aortic valves, is the strain of prolonged and heavy muscular exertion. In no other way can be explained the occurrence of so many cases of sclero-

sis of the aortic valves in young and middle-aged men whose occupations necessitate the overuse of the muscles.

Morbid Anatomy.—Vegetations in the form in which they occur in acute endocarditis are not present. In the early stage, which we have frequent opportunities of seeing, the edge of the valve is a little thickened and perhaps presents a few small nodular prominences, which in some cases may represent the healed vegetations of the acute process. In the aortic valves the tissue about the corpora Arantii is first affected, producing a slight thickening with an increase in the size of the nodules. The substance of the valve may lose its translucency, and the only change noticeable is a grayish opacity and a slight loss of its delicate tenuity. In the auriculo-ventricular valves these early changes are seen just within the margin and here it is not uncommon to find swellings of a grayish-red, somewhat infiltrated appearance, almost identical with the similar structures on the intima of the aorta in arterio-sclerosis. Even early there may be seen yellow or opaque-white subintimal fatty areas. As the sclerotic changes increase the fibrous tissue contracts and produces thickening and deformity of the segment, the edges of which become round, curled, and incapable of that delicate apposition necessary for perfect closure. A sigmoid valve, for instance, may be narrowed one fourth or even one third across its face, inducing the most extreme grade of insufficiency without any special deformity and without any definite narrowing of the arterial orifice. In the auriculo-ventricular segments a simple process of thickening and curling of the edges of the valves, inducing a failure to close without forming any obstruction to the normal course of the blood-flow, is less common. Still, we meet with instances at the mitral orifice, particularly in children, in which the edges of the valves are curled and thickened, producing extreme insufficiency without any material narrowing of the orifice. More frequently, as the disease advances, the chordæ tendineæ become thickened, first at the valvular ends and then along their course. The edges of the valves at their angles are gradually drawn together and there is a definite narrowing of the orifice, leading in the aorta to more or less stenosis and in the left auriculo-ventricular orifice—the two most frequently involved—to constriction. Finally, in the sclerotic and necrotic tissues lime salts are deposited and may even reach the deeper structures of the fibrous rings, and the entire valve becomes a dense calcareous mass with scarcely a remnant of normal tissue. The chordæ tendineæ may gradually become shortened, greatly thickened, and in extreme cases the papillary muscles are implanted directly upon the sclerotic and deformed valve. The apices of the papillary muscles usually show marked fibroid change.

In all stages of the process the vegetations of simple endocarditis may be found and upon sclerotic valves we find the severer, ulcerative form of the disease.

Chronic *mural* endocarditis produces cicatricial-like patches of a gray-

ish-white appearance which are sometimes seen on the muscular trabeculæ of the ventricle or in the auricles. It often occurs in association with myocarditis.

The frequency with which chronic endocarditis is met with may be gathered from the following figures: In the statistics, amounting to from 12,000 to 14,000 autopsies, reported from Dresden, Wurzburg, and Prague the percentage ranged from four to nine. The relative frequency of involvement of the various valves is thus given in the collected statistics of Parrot: The mitral orifice was involved in 621, the aortic in 380, the tricuspid in 46, and the pulmonary in 11. This gives 57 instances in the right to 1,001 in the left heart.

The endocarditis of the fœtus is usually of the sclerotic form and involves the valves of the right more frequently than those of the left side.

The effects of sclerotic endocarditis are practically those of chronic valvular disease, and the general influence on the work of the heart may be briefly stated as follows: The sclerosis induces insufficiency or stenosis, which may exist separately or in combination. The narrowing retards in a measure the normal outflow and the insufficiency permits the blood current to take an abnormal course. In both instances the effect is dilatation of a chamber. The result in the former case is an increase in the difficulty which the chamber has in expelling its contents through the narrow orifice; in the other, the overfilling of a chamber by blood flowing into it from an improper source, as, for instance, in mitral insufficiency, when the left auricle receives blood both from the pulmonary veins and from the left ventricle.

The cardiac mechanism is fully prepared to meet ordinary grades of dilatation which constantly occur during sudden exertion. A man, for instance, at the end of a hundred-yard race has his right chambers greatly dilated and his reserve cardiac power worked to its full capacity. The slow progress of the sclerotic changes brings about a gradual, not an abrupt, insufficiency, and the moderate dilatation which follows is at first overcome by the exercise of the ordinary reserve strength of the heart muscles. Gradually a new factor is introduced. The reserve power which is capable of meeting sudden emergencies in such a remarkable manner is unable to cope long with a permanent and perhaps increasing dilatation. More work has to be done and, in accordance with definite physiological laws, more power is given by increase of the muscles. The heart hypertrophies and the effect of the valve lesion becomes, as we say, *compensated*. The equilibrium of the circulation is in this way maintained.

II. CHRONIC VALVULAR DISEASE.

Aortic Incompetency.

Incompetency of the aortic valves arises either from inability of the valve segments to close an abnormally large orifice or more commonly from disease of the segments themselves. This best-defined and most easily recognized of valvular lesions was first carefully studied by Corrigan, whose name it sometimes bears.

Etiology and Morbid Anatomy.—It is more frequent in males than in females, affecting chiefly able-bodied, vigorous men at the middle period of life. The ratio which it bears to other valve diseases has been variously given from thirty to fifty per cent.

Among the important factors in producing this condition are: (*a*) Congenital malformation, particularly fusion of two segments—most commonly those behind which the coronary arteries are given off. It is probable that an aortic orifice may be competent with this bicuspid state of the valves, but a great danger is the liability of these malformed segments to sclerotic endocarditis. Of seventeen cases which I have reported all presented sclerotic changes, and the majority of them had, during life, the clinical features of chronic heart-disease.

(*b*) Acute endocarditis. This does not produce aortic incompetency unless the process passes on to ulceration and destruction, under which circumstances it is often found, and may cause a rapidly fatal issue. Simple endocarditis associated with the specific fevers is not nearly so common on the aortic as on the mitral segments; so also with rheumatism, which plays a less important *rôle* here than in mitral valve disease.

(*c*) By far the most frequent cause of insufficiency is the slow, progressive sclerosis of the segment, resulting in a curling of the edge, which lessens the working surface of the valve. This may, of course, follow acute endocarditis, but it is so often met with in strong, able-bodied men among the working classes, without any history of rheumatism or special febrile diseases with which endocarditis is commonly associated, that other conditions must be sought for to explain its frequency. Of these, unquestionably strain is the most important—not a sudden, forcible strain, but a persistent increase of the normal tension to which the segments are subject during the diastole of the ventricle. Of circumstances increasing this tension, heavy and excessive use of the muscles is perhaps the most important. So often is this form of heart-disease found in persons devoted to athletics that it is sometimes called the "athlete's heart." Alcohol is a second important factor, and is stated to raise considerably the tension in the aortic system. A combination of these two causes is extremely common. A third element in inducing chronic sclerotic changes in these valves is syphilis. Cases are rarely seen in which other factors must not be taken into account, but the association is too

frequent to be accidental. That syphilis is capable of inducing arterial sclerosis is, I think, acknowledged, although the way in which it is done is not yet clear. It is interesting to note with what frequency this form of valve disease occurs in soldiers. I was struck with this fact in the Philadelphia Hospital, to which so many veterans of the civil war are admitted. I was in the habit of enforcing upon my students the etiological lesson by a mythological reference to Bacchus and Vulcan, at whose shrines a majority of the cases of aortic insufficiency have worshipped, and not a few at that of Venus.

The condition of the valves is such as has already been described in chronic endocarditis. It may be noted, however, how slight a grade of curling may produce serious incompetency. Associated with the valve disease is, in a majority of the cases, a more or less advanced arterio-sclerosis of the arch of the aorta, one serious effect of which may be a narrowing of the orifices of the coronary arteries. The sclerotic changes are often combined with atheroma, either in the fatty or calcareous stage. This may exist at the attached margin of the valves without inducing insufficiency. In other instances insufficiency may result from a calcified spike projecting from the aortic attachment into the body of the valve, and so preventing its proper closure. Some writers (Peter) have laid great stress upon the extension of the endarteritis to the valve, and would separate the instances of this kind from those of simple valvular endocarditis. I must say that I have not been able to recognize clinical differences between these two conditions, though anatomically we may separate the cases into two groups—those with and those without arterio-sclerosis.

(*d*) And, lastly, insufficiency may be induced by rupture of a segment —a very rare event in healthy valves, but not uncommon in disease, either from excessive strain during heavy lifting or from the ordinary endarterial strain in a valve eroded and weakened by ulcerative endocarditis.

Relative insufficiency of the sigmoid valves, due to dilatation of the aortic ring, is a rare condition. It is said to occur in extensive arterial sclerosis of the ascending portion of the arch with great dilatation just above the valves. I have myself never met with a pure instance of the kind, for in such cases I have always found the valve segments involved with the arterial coats. In aneurism just above the aortic ring, relative insufficiency of the valve may be present.

It would appear from the careful measurements of Beneke that the aortic orifice, which at birth is 20 mm., increases gradually with the growth of the heart until at one and twenty it is about 60 mm. At this it remains until the age of forty, beyond which date there is a gradual increase in the size up to the age of eighty, when it may reach from 68 to 70 mm. There is thus at the very period of life in which sclerosis of the valve is most common a physiological tendency toward the production of a state of relative insufficiency.

The insufficiency may be combined with various grades of narrowing, but the majority of the cases of aortic insufficiency present no signs of stenosis. On the other hand, cases of aortic stenosis almost without exception are associated with some grade, however slight, of regurgitation.

The direct effect of aortic insufficiency is the regurgitation of blood from the artery into the ventricle, causing an overdistention of the cavity and a reduction of the blood column; that is, a relative anæmia in the arterial tree. As an immediate effect of the double blood-flow into the left ventricle dilatation of the chamber occurs, and finally hypertrophy. In this way the valve defect is compensated and as with each ventricular systole a larger amount of blood is propelled into the arterial system, the regurgitation of a certain amount during diastole does not, for a time at least, seriously impair the nutrition of the peripheral parts. In this valve lesion dilatation and hypertrophy reach their most extreme limit. The heaviest hearts on record are described in connection with this affection. The so-called bovine heart, *cor bovinum*, may weigh 35 or 40 ounces, or even, as in a case of Dulles's, 48 ounces. The dilatation is usually extreme, and is in marked contrast to the condition of the chamber in cases of pure aortic stenosis. The papillary muscles may be greatly flattened. The mitral valves are usually not seriously affected, though the edges may present slight sclerosis, and there is often relative incompetency, owing to distention of the mitral ring. Dilatation and hypertrophy of the left auricle are common, and secondary enlargement of the right heart occurs in all cases of long standing. The myocardium usually presents changes, fibroid or fatty; more commonly the former in association with disease of the coronary arteries. The arch of the aorta may present extensive arterio-sclerosis and dilatation. In rare instances, usually the rheumatic cases, the intima is perfectly smooth, and the arch with its main branches not dilated. This condition may be found post mortem even when during life there have been the most characteristic signs of enlargement of the arch and of dilatation of the innominate and right carotid. I have even known the condition of aneurism to be diagnosed when post mortem no trace of dilatation or sclerosis was found, only an extreme grade of insufficiency with enormous dilatation and hypertrophy. The coronary arteries are usually involved in the sclerosis, and their orifices may be much narrowed. Although these vessels have been shown by Martin and Sedgwick to be filled during the ventricular systole, the circulation in them must be embarrassed in aortic incompetency. They must miss the effect of the blood-pressure in the sinuses of Valsalva during the elastic recoil of the arteries, which surely aids in keeping the coronary vessels full. The arteries of the body usually present more or less sclerosis consequent upon the strain which they undergo during the forcible ventricular systole.

Symptoms.—The condition is often discovered accidentally in persons who have not presented any features of cardiac disease.

Physical Signs.—*Inspection* shows a wide and forcible area of cardiac impulse with the apex beat in the sixth or seventh interspace, and perhaps as far out as the anterior axillary line. In young subjects the præcordia may bulge. On palpation a thrill, diastolic in time, is occasionally felt, but is not common. The impulse is usually strong and heaving, unless in conditions of extreme dilatation, when it is wavy and indefinite. *Percussion* shows a greater increase in the area of heart dulness than is found in any other valvular lesion. It extends chiefly downward and to the left.

On *auscultation* there is heard a murmur during diastole in the second right interspace, which is propagated with intensity toward the ensiform cartilage or down the left margin of the sternum toward the apex. In the majority of cases it is a soft, long-drawn *bruit*, and is of all cardiac murmurs the most reliable. It occurs during the time of, and is produced by, the reflux of blood from the aorta into the ventricle. In a large proportion of the cases there is also a systolic murmur heard at the aortic region, usually shorter, often rougher in quality, and which may be propagated upward into the neck. A common mistake is to regard this as indicating stenosis, whereas in the great majority of instances of aortic insufficiency there is no material narrowing, and the murmur is produced by roughening of the segments or of the intima of the arch. The second sound is usually obliterated, though in some instances both the murmur and the valvular sound may be distinctly heard. At the apex murmurs are also heard, either transmitted from the aortic orifice or produced at the mitral. In the majority of cases with aortic incompetency of high grade, the mitral orifice is dilated, and there is relative insufficiency of the valves. It can frequently be determined that the systolic murmur at the apex differs in quality from that at the base. A second murmur at the apex, probably produced at the mitral orifice, is not infrequent. Attention was called to this by the late Austin Flint, and the murmur usually goes by his name. It has a distinctly rumbling quality, is limited in area, and is sometimes, though not always, distinctly presystolic in time. The explanation of its occurrence, as given by Flint, is that in the extreme dilatation of the ventricle the mitral segments cannot during diastole be forced back against the wall, and, therefore, remaining in the blood current, they produce a sort of relative narrowing, and in consequence a vibratory murmur not unlike in quality the presystolic murmur of mitral stenosis. My experience as to the frequency of this murmur coincides with that of Lee.*

The examination of the arteries in aortic insufficiency is of great value. Visible pulsation is more commonly seen in the peripheral vessels in this than in any other condition. The carotids may be seen to throb forcibly, the temporals to dilate, and the brachials and radials to expand with each

* American Journal of the Medical Sciences, 1890.

heart-beat. With the ophthalmoscope the retinal arteries are seen to pulsate. Not only is the pulsation evident, but the characteristic jerking quality is apparent. In the throat the throbbing carotids may lead to the diagnosis of aneurism. In many cases the pulsation can be seen in the suprasternal notch, and prominent, forcibly-throbbing vessels beneath the right sterno-mastoid muscle. The abdominal aorta may lift the epigastrium with each systole. To be mentioned with this is the capillary pulse, met very often in aortic insufficiency, and best seen in the finger-nails or by drawing a line upon the forehead, when the margin of hyperæmia on either side alternately blushes and pales. In extreme grades the face or the hand may blush visibly at each systole. It is met with also in profound anæmia, occasionally in neurasthenia, and in health in conditions of great relaxation of the peripheral arteries. Pulsation may also be present in the peripheral veins. On palpation the characteristic water-hammer or Corrigan pulse is felt. On the majority of instances the pulse wave strikes the finger forcibly with a quick jerking impulse, and immediately recedes or collapses. The characters of this are sometimes best appreciated by grasping the arm above the wrist and holding it up. On auscultation a double murmur may be heard in the carotids and subclavians when it is present at the aortic orifice. Occasionally in the carotid the second sound is distinctly audible when absent at the aortic cartilage. In the femoral artery a double murmur also may be heard sometimes, as pointed out by Duroziez.

Aortic insufficiency may for years be fully compensated. Persons do not necessarily suffer any inconvenience, and the condition is often found accidentally. So long as the hypertrophy just equalizes the valvular defect there may be no symptoms and the individual may even take moderately heavy exercise without experiencing sensations of distress about the heart. The cases which last the longest are those in which the sclerosis follows endocarditis and is not a part of a general arterio-sclerosis. Coexistent lesions of the mitral valves tend early to disturb the compensation. It has scarcely been sufficiently recognized by the profession at large that pure aortic insufficiency is consistent with years of average health and with a tolerably active life. I know several physicians with aortic insufficiency who have been able to carry on for years large and somewhat onerous practices. One of them since the establishment of insufficiency has passed successfully through two attacks of acute rheumatism. In large hospital practice, scarcely a month passes without the discovery of a case of aortic insufficiency in connection with some other affection.

With the onset of myocardial changes, with increasing degeneration of the arteries, particularly with a progressive sclerosis of the arch and involvement of the orifices of the coronary arteries, the compensation becomes disturbed. In advanced cases the changes about the aortic ring may be associated with alterations in the cardiac nerves and ganglia, and so introduce an important factor.

Headache, dizziness, flashes of light, and a feeling of faintness on rising quickly are among the earliest symptoms. Palpitation and cardiac distress on slight exertion are common. Long before any signs of failing compensation pain may become a marked and troublesome feature. It is extremely variable in its manifestations. It may be of a dull, aching character confined to the præcordia. More frequently, however, it is sharp and radiating, and is transmitted up the neck and down the arms, particularly the left. Attacks of true angina pectoris are more frequent in this than in any other valvular disease. Anæmia is also common, much more so than in aortic stenosis or in mitral affections.

More serious symptoms, as compensation fails, are shortness of breath and œdema of the feet. The attacks of dyspnœa are liable to come on at night and the patient has to sleep with the head high or even in a chair. Of respiratory symptoms cough may develop, due to the congestion of the lungs or œdema. Hæmoptysis is less frequent than in mitral disease. I have reported a case in which it was profuse and believed to be due to tuberculosis of the lungs, inasmuch as the patient was admitted in a state of emaciation and profound exhaustion. General dropsy is not common, but œdema of the feet may occur early and is sometimes due to the anæmia, at others to the venous stasis, at times to both. Unless there is coexisting disease of the mitral valve, it is rare in pure aortic incompetency for the patient to die with general anasarca. Sudden death is frequent; more so in this than in other valvular diseases. As compensation fails the patient takes to bed and slight irregular fever, associated usually with a recurring endocarditis, is not uncommon toward the close. Embolic symptoms are not infrequent—pain in the splenic region with enlargement of the organ, hæmaturia, and in some cases paralysis. Distressing dreams and disturbed sleep are more common in this than in other forms of valvular disease.

Here may appropriately be mentioned the connection between mental symptoms and cardiac disease, as they are oftenest seen with this lesion. An admirable account of the relations between insanity and disease of the heart is to be found in Mickle's Gulstonian lectures for 1888. In general medical practice we seldom find marked mental symptoms, except toward the close of the disease, when there may be delirium, hallucinations, and morbid impulses. It is to be remembered that in many heart cases this terminal delirium is uræmic. The irritability and peevishness sometimes found in persons the subject of organic heart-disease cannot, I think, be associated with it in any special manner. We do meet insanity, breaking out in patients with aortic and mitral disease, in the stage of compensation, which appears to be related definitely to the cardiac lesion. It is important to bear this in mind, for cases occasionally display suicidal tendencies. I have twice had patients throw themselves from the window of the ward.

Aortic Stenosis.

Narrowing or stricture of the aortic orifice is not nearly so common as insufficiency. The two conditions, as already stated, may occur together, however, and probably in almost every case of stenosis there is some leakage.

Etiology and Morbid Anatomy.—In the milder grades there is adhesion between the segments, which are so stiffened that during systole they cannot be pressed back against the aortic wall. The process of cohesion between the segments may go on without great thickening, and produce a condition in which the orifice is guarded by a comparatively thin membrane, on the aortic face of which may be seen the primitive raphés separating the sinuses of Valsalva. In some instances this membrane is so thin and presents so few traces of atheromatous or sclerotic changes that the condition looks as if it had originated during fœtal life. More commonly the valve segments are thickened and rigid, and have a cartilaginous hardness. In advanced cases they may be represented by stiff, calcified masses obstructing the orifice, through which a circular or slit-like passage can be seen. The older the patient the more likely it is that the valves will be rigid and calcified.

We may speak of a relative stenosis of the aortic orifice when with normal valves and ring the aorta immediately beyond is greatly dilated. A stenosis due to involvement of the aortic ring in sclerotic and calcareous changes without lesion of the valves is referred to by some authors. I have never met with an instance of this kind. A subvalvular stenosis, the result of endocarditis in the mitro-sigmoidean sinus, usually occurs as the result of a fœtal endocarditis. In comparison with aortic insufficiency, stenosis is a rare disease. It is usually met with at a more advanced period of life than insufficiency, and the most typical cases of it are found associated with extensive calcareous changes in the arterial system in old men.

When gradually produced and when there is not much insufficiency the dilatation of the left ventricle may be slight, though I think that in all cases it does occur. The walls of the ventricle become hypertrophied, and we see in this condition the most typical instances of what is called concentric hypertrophy, in which, without much, if any, enlargement of the cavity, the walls are greatly thickened, in contradistinction to the so-called eccentric hypertrophy, in which the chamber is greatly dilated as well as hypertrophied. There may be no changes in the other cardiac cavities if compensation is well maintained; but with its failure come dilatation, impeded auricular discharge, pulmonary congestion, and increased work for the right heart. The arterial changes are, as a rule, not so marked as in aortic insufficiency, for the walls have not to withstand the impulse of a greatly increased blood-wave with each systole. On the contrary, the amount of blood propelled through the narrow orifice may be smaller than normal, though when compensation is fully established the pulse-wave may be of medium volume.

Symptoms. Physical Signs.—*Inspection* may fail to reveal any area of cardiac impulse. Particularly is this the case in old men with rigid chest walls and large emphysematous lungs. Under these circumstances there may be a high grade of hypertrophy without any visible impulse. Even when the apex beat is visible it may be, as Traube pointed out, feeble and indefinite. In many cases the apex is seen displaced downward and outward, and the impulse looks strong and forcible.

Palpation reveals in many cases a thrill at the base of the heart of maximum force in the aortic region. With no other condition do we meet with thrills of greater intensity. The apex beat may not be palpable under the conditions above mentioned, or there may be a slow, heaving, forcible impulse.

Percussion never gives the same wide area of dulness as in aortic insufficiency. The extent of it depends largely on the state of the lungs, whether emphysematous or not.

Auscultation.—A systolic murmur of maximum intensity at the aortic cartilage, and propagated into the great vessels, is present in aortic stenosis, but is by no means pathognomonic. One of the last lessons learned by the student of physical diagnosis is to recognize the fact that this systolic murmur is only in comparatively rare cases produced by decided narrowing of the aortic orifice. Roughening of the valves, or the intima of the aorta, and hæmic states are much more frequent causes. In aortic stenosis the murmur often has a much harsher quality, is louder, and is more frequently musical than in the conditions just mentioned. When compensation fails and the ventricle is dilated and feeble the murmur may be soft and distant. The second sound is rarely heard at the aortic cartilage, owing to the thickening and stiffness of the valve. A diastolic murmur is not uncommon, but in many cases it cannot be heard. The pulse in pure aortic stenosis is small, usually of good tension, regular, and perhaps slower than normal.

The condition may be latent for an indefinite period, as long as the hypertrophy is maintained. Early symptoms are those due to defective blood-supply to the brain, dizziness, and fainting. Palpitation, pain about the heart, and anginal symptoms are not so marked as in insufficiency. With degeneration of the heart-muscle and dilatation relative insufficiency of the mitral valve is established, and the patient may present all the features of engorgement in the lesser and systemic circulations, with dyspnœa, cough, rusty expectoration, and the signs of anasarca in the lower part of the body. Many of the cases in old people, without presenting any dropsy, have symptoms pointing rather to general arterial disease. Cheyne-Stokes breathing is not uncommon with or without signs of uræmia.

Diagnosis.—With an intensely rough or musical murmur of maximum intensity at the aortic region and signs of hypertrophy of the left ventricle, a thrill and a hard, slow pulse of moderate volume and fairly good tension,

a diagnosis of aortic stenosis can be made with some degree of probability, particularly if the subject is an old man. Mistakes are common, however, and a roughened or calcified valve segment, or, in some instances, a very roughened and prominent calcified plate in the aorta, and hypertrophy associated with renal disease, may produce similar symptoms.

Let me repeat that a murmur of maximum intensity at the aortic cartilage is of no importance in itself as a diagnostic sign of stenosis. Roughening of the valve, sclerosis of the intima of the arch, and anæmia are conditions more frequently associated with a systolic murmur in this region. Seldom is there difficulty in distinguishing the murmur due to anæmia, since it is rarely so intense and is not associated with thrill or with marked hypertrophy of the left ventricle. In aortic insufficiency a systolic murmur is usually present, but has neither the intensity nor the musical quality, nor is it accompanied with a thrill. With roughening and dilatation of the ascending aorta the murmur may be very harsh or musical; but the existence of a second sound, accentuated and ringing in quality, is usually sufficient to differentiate this condition.

Mitral Incompetency.

Etiology. — Insufficiency of the mitral valve results from: (*a*) Changes in the segments whereby they are contracted and shortened, usually combined with changes in the chordæ tendineæ, or with more or less narrowing of the orifice. (*b*) As a result of changes in the muscular walls of the ventricle, either dilatation, so that the valve segments fail to close an enlarged orifice, or changes in the muscular substance, so that the segments are imperfectly coapted during the systole—muscular incompetency. The common lesions producing insufficiency result from endocarditis, which causes a gradual thickening at the edges of the valves, contraction of the chordæ tendineæ, and union of the edges of the segments, so that in a majority of the instances there is not only insufficiency, but some grade of narrowing as well. Except in children, we rarely see the mitral leaflets curled and puckered without narrowing of the orifice. Calcareous plates at the base of the valve may prevent perfect closure of one of the segments. In long-standing cases the entire mitral structures are converted into a firm calcareous ring. From this valvular insufficiency the other condition of muscular incompetency must be carefully distinguished. It is met with in all conditions of extreme dilatation of the left ventricle, and also in weakening of the muscles in prolonged fevers and in anæmia.

Morbid Anatomy.—The effects of incompetency of the mitral segment upon the heart and circulation are as follows: (*a*) The imperfect closure allows a certain amount of blood to regurgitate from the ventricle into the auricle, so that at the end of auricular diastole this chamber con-

tains not only the blood which it has received from the lungs, but also that which has regurgitated from the left ventricle. This necessitates dilatation, and, as increased work is thrown upon it in expelling the augmented contents, hypertrophy as well.

(*b*) With each systole of the left auricle a larger volume of blood is forced into the left ventricle, which also dilates and subsequently becomes hypertrophied.

(*c*) During the diastole of the left auricle, as blood is regurgitated into it from the left ventricle, the pulmonary veins are less readily emptied. In consequence the right ventricle expels its contents less freely, and in turn becomes dilated and hypertrophied.

(*d*) Finally, the right auricle also is involved, its chamber is enlarged, and its walls are increased in thickness.

(*e*) The effect upon the pulmonary vessels is to produce dilatation both of the arteries and veins—often in long-standing cases atheromatous changes; the capillaries are distended, and ultimately the condition of brown induration is produced. Perfect compensation may be effected, chiefly through the hypertrophy of both ventricles, and the effect upon the peripheral circulation may not be manifested for years, as a normal volume of blood is discharged from the left heart at each systole. The time comes, however, when, owing either to increase in the grade of the incompetency or to failure of the compensation, the left ventricle is unable to send out its normal volume into the aorta. Then there is overfilling of the left auricle, engorgement in the lesser circulation, embarrassed action of the right heart, and congestion in the systemic veins. For years this somewhat congested condition may be limited to the lesser circulation, but finally the right auricle becomes dilated, the tricuspid valves incompetent, and the systemic veins are engorged. This gradually leads to the condition of cyanotic induration in the viscera and, when extreme, to dropsical effusion.

Muscular incompetency, due to impaired nutrition of the mitral and papillary muscles, is rarely followed by such perfect compensation. There may be in acute destruction of the aortic segments an acute dilatation of the left ventricle with relative incompetency of the mitral segments, great dilatation of the left auricle, and intense engorgement of the lungs, under which circumstances profuse hæmorrhage may result. In these cases there is little chance for the establishment of compensation. In cases of hypertrophy and dilatation of the heart, without valvular lesions, but associated with heavy work and alcohol, the insufficiency of the mitral valve may be extreme and lead to great pulmonary congestion, engorgement of the systemic veins, and a condition of cardiac dropsy, which cannot be distinguished by any feature from that of mitral incompetency due to lesion of the valve itself. In chronic Bright's disease the hypertrophy of the left ventricle may gradually fail, leading, in the later stages, to relative insufficiency of the mitral valve, and the production of a con-

dition of pulmonary and systemic congestion, similar to that induced by the most extreme grade of lesion of the valve itself.

Symptoms.—During the development of the lesion, unless the incompetency comes on acutely in consequence of rupture of the valve segment or of ulceration, the compensatory changes go hand in hand with the defect, and there are no subjective symptoms. So, also, in the stage of perfect compensation, there may be the most extreme grade of mitral insufficiency with enormous hypertrophy of the heart, yet the patient may not be aware of the existence of heart trouble, and may suffer no inconvenience except perhaps a little shortness of breath on exertion or on going up-stairs. It is only when from any cause the compensation has not been perfectly effected, or having been so is broken abruptly or gradually, that the patients begin to be troubled. The symptoms may be divided into two groups:

(*a*) The minor manifestations while compensation is still good. Patients with extreme incompetency often have a congested appearance of the face, the lips and ears have a bluish tint, and the venules on the cheeks may be enlarged, which in many cases is very suggestive. In long-standing cases, particularly in children, the fingers may be clubbed, and there is shortness of breath on exertion. This is one of the most constant features in mitral insufficiency, and may exist for years, even when the compensation is perfect. Owing to the somewhat congested condition of the lungs these patients have a tendency to attacks of bronchitis or hæmoptysis. There may also be palpitation of the heart. As a rule, however, in well-balanced lesions in adults, this period of full compensation or latent stage is not associated with symptoms which call the attention of the patient to an affection of the heart.

(*b*) Sooner or later comes a period of disturbed or broken compensation, in which the most intense symptoms are those of venous engorgement. There are palpitation, weak, irregular action of the heart, and signs of dilatation. Dyspnœa is a marked feature, and there may be cough. There is usually a slight cyanosis, and even a jaundiced tint to the skin. The most marked symptoms, however, are those of venous stasis. The overfilling of the pulmonary vessels accounts in part for the dyspnœa. There is cough, often with bloody or watery expectoration, and the alveolar epithelium containing brown pigment-grains is abundant. Dropsical effusion usually sets in, beginning in the feet and extending to the body and the serous sacs. The liver is enlarged, and there are signs of portal congestion, gastric irritation, and catarrh of the stomach and intestines. The urine is usually scanty and albuminous, and contains tube casts and sometimes blood-corpuscles. With judicious treatment the compensation may be restored and all the serious symptoms may pass away. Patients may have recurring attacks of this kind, but ultimately the condition is beyond repair and the patient either dies of a general

dropsy or there is progressive dilatation of the heart, and death from asystole. Sudden death in these cases is rare.

Physical Signs.—*Inspection.*—In children the præcordia may bulge and there may be a large area of visible pulsation. The apex beat is to the left of the nipple, in some cases in the sixth interspace, in the anterior axillary line. There may be a wavy impulse in the cervical veins which are often full, particularly when the patient is recumbent.

Palpation.—A thrill is rare; when present it is felt at the apex, often in a limited area. The force of the impulse may depend largely upon the stage in which the case is examined. In full compensation it is forcible and heaving; when the compensation is disturbed, usually wavy and feeble.

Percussion.—The dulness is increased, particularly in a lateral direction. There is no disease of the valves which produces, in long-standing cases, a more extensive transverse area of heart dulness. It does not extend so much upward along the left margin of the sternum as beyond the right margin and to the left of the nipple line.

Auscultation.—At the apex there is a systolic murmur which wholly or partly obliterates the first sound. It is loudest here, and has a blowing, sometimes musical in character, particularly toward the latter part. The murmur is transmitted to the axilla and may be heard at the back, in some instances over the entire chest. There are cases in which, as pointed out by Naunyn, the murmur is heard best along the left border of the sternum. Usually in diastole at the apex the loudly transmitted second sound may be heard. Occasionally there is also a soft, sometimes a rough or rumbling presystolic murmur. As a rule, in cases of extreme mitral insufficiency from valvular lesion with great hypertrophy of both ventricles, there is heard only a loud blowing murmur during systole. A murmur of mitral insufficiency may vary a great deal according to the position of the patient. It may be present in the recumbent and absent in the erect posture. In cases of dilatation, particularly when dropsy is present, there may be heard at the ensiform cartilage and in the lower sternal region a soft systolic murmur due to tricuspid regurgitation. An important sign on auscultation is the accentuated pulmonary second sound. This is heard to the left of the sternum in the second interspace, or over the third left costal cartilage.

The pulse in mitral insufficiency, during the period of full compensation, may be full and regular, often of low tension. Usually with the first onset of the symptoms the pulse becomes irregular, a feature which then dominates the case throughout. There may be no two beats of equal force or volume. Often after the disappearance of the symptoms of failure of compensation the irregularity of the pulse persists.

The three important physical signs then of mitral regurgitation are: (*a*) systolic murmur of maximum intensity at the apex, which is propagated to the axilla and heard at the angle of the scapula; (*b*) accentuation

of the pulmonary second sound; (*c*) evidence of enlargement of the heart, particularly the increase in the transverse diameter, due to hypertrophy of both right and left ventricles.

Diagnosis.—There is rarely any difficulty in the diagnosis of mitral insufficiency. The physical signs just referred to are quite characteristic and distinctive. Two points are to be borne in mind. First, a murmur, systolic in character, and of maximum intensity at the apex, and propagated even to the axilla, does not necessarily indicate incompetency of the mitral valve. There is heard in this region a large group of what are termed accidental murmurs, the precise nature of which is still doubtful. They are probably formed, however, in the ventricle, and are not associated with hypertrophy, or accentuation of pulmonary second sound.

Second, it is not always possible to say whether the insufficiency is due to lesion of the valve segment or to dilatation of the mitral ring and relative incompetency. Here neither the character of the murmur, the propagation, the accentuation of the pulmonary second sound, nor the hypertrophy assists in the differentiation. The history is sometimes of greater value in this matter than the physical examination. The cases most likely to lead to error are those of the so-called idiopathic dilatation and hypertrophy of the heart (in which the systolic murmur may be of the greatest intensity), and the instances of arterio-sclerosis with dilated heart.

Mitral Stenosis.

Etiology.—Narrowing of the mitral orifice is usually the result of valvular endocarditis occurring in the earlier years of life; very rarely it is congenital. It is very much more common in women than in men—in 63 of 80 cases noted by Duckworth. This is not easy to explain, but there are at least two factors to be considered. Rheumatism prevails more in girls than in boys and, as is well known, endocarditis of the mitral valve is more common in rheumatism. Chorea, also, as suggested by Barlow, has an important influence, occurring more frequently in girls and often associated with endocarditis. Of 110 cases of chorea which I examined at a period more than two years subsequent to the attack, 54 cases had signs of organic heart-disease, among which were 17 instances with the physical signs of mitral stenosis. Anæmia and chlorosis, which are prevalent in girls, have been regarded as possible factors. In a number of cases, however, no recognizable etiological factor can be discovered. This has been regarded by some writers as favoring the view that many of the cases are of congenital origin; but it is not improbable that with any of the febrile affections of childhood endocarditis may be associated. Whooping-cough, too, with its terrible strain on the heart-valves, may be accountable for certain cases. Congenital affections of the mitral valve are notoriously rare. While met with at all ages, stenosis is certainly more frequent in young persons.

Morbid Anatomy.—In a majority of instances with the stenosis there is some incompetency. The narrowing results from thickening and contraction of the tissues of the ring, of the valve segments, and of the chordæ tendineæ. The condition varies a good deal according to the amount of atheromatous change. In many cases the curtains are so welded together and the whole valvular region so thickened that the orifice is reduced to a mere chink—Corrigan's button-hole contraction. In other cases the curtains are not much thickened, but narrowing has resulted from gradual adhesion at the edges, and thickening of the chordæ tendineæ, so that from the auricle it looks cone-like—the so-called funnel-shaped variety of stenosis. The instances in which the valve segments are very slightly deformed but in which the orifice is considerably narrowed, are regarded by some as possibly of congenital origin. Occasionally the curtains are in great part free from disease, but the narrowing results from large calcareous masses, which project into them from the ring. The involvement of the chordæ tendineæ is usually extreme, and the papillary muscles may be inserted directly upon the valve. In moderate grades of constriction the orifice will admit the tip of the index-finger; in more extreme forms, the tip of the little finger; and occasionally one meets with a specimen in which the orifice seems almost obliterated, as in a case which came under my notice, which only admitted a medium-sized Bowman's probe.

The heart in mitral stenosis is not greatly enlarged, rarely weighing more than 14 or 15 ounces. Occasionally, in an elderly person, it may seem slightly if at all enlarged, and again there are instances in which the weight may reach as much as 20 ounces. The left ventricle is usually small, and may look very small in comparison with the right ventricle, which forms the greater portion of the apex. In cases in which with the narrowing there is very considerable incompetency the left ventricle may be moderately dilated and hypertrophied.

These changes gradually induced are associated with secondary alterations of great importance in the heart. The left auricle discharges its blood with greater difficulty and in consequence dilates, and its walls reach three or four times their normal thickness. Although the auricle is by structure unfitted to compensate an extreme lesion, the probability is that for some time during the gradual production of stenosis, the increasing muscular power of the walls is sufficient to counterbalance the defect. Eventually the tension is increased in the pulmonary circulation, owing to impeded outflow from the veins. To overcome this the right ventricle undergoes dilatation and hypertrophy, and upon this chamber falls the work of equalizing the circulation. Relative incompetency of the tricuspid and congestion of systemic veins at last supervene.

It is not uncommon at the examination to find white thrombi in the appendix of the left auricle. Occasionally a large part of the auricle is occupied by an ante-mortem thrombus. Still more rarely the remarkable

ball thrombus is found, in which a globular concretion, varying in size from a walnut to a small egg, lies free in the auricle, two examples of which have come under my observation.

Symptoms.—Physical Signs.—*Inspection.*—In children the lower sternum and the fifth and sixth left costal cartilages are often prominent, owing to hypertrophy of the right ventricle. The apex beat may be ill-defined. Usually, it is not dislocated far beyond the nipple line, and the chief impulse is over the lower sternum and adjacent costal cartilages. Often in thin-chested persons there is pulsation in the third and fourth left interspaces close to the sternum. When compensation fails, the præcordial impulse is much feebler, and in the veins of the neck there may be marked systolic regurgitation.

Palpation reveals in a majority of the cases a characteristic, well-defined fremitus or thrill, which is best felt, as a rule, in the fourth or fifth interspace within the nipple line. It is of a rough, grating quality, often peculiarly limited in area, most marked during expiration, and can be felt to terminate in a sharp, sudden shock, synchronous with the impulse. This most characteristic of physical signs is pathognomonic of narrowing of the mitral orifice, and is perhaps the only instance in which the diagnosis of a valvular lesion can be made by palpation alone. The cardiac impulse is felt most forcibly in the lower sternum and in the fourth and fifth left interspaces. The impulse is felt very high in the third and fourth interspaces, or in rare cases even in the second, and it has been thought that in the latter interspace the impulse is due to pulsation of the auricle. It is always the impulse of the right ventricle; even in the most extreme grades of mitral stenosis, there is never such tilting forward of the auricle or its appendix as would enable it to produce an impression on the chest wall.

Percussion gives an increase in the cardiac dulness to the right of the sternum and along the left margin; not usually a great increase beyond the nipple line, except in extreme cases, when the transverse dulness may reach from 5 cm. beyond the right margin of the sternum to 10 cm. beyond the nipple line.

Auscultation.—In the mitral area, usually to the inner side of the apex beat and often in a very limited region, is heard a rough, vibratory or purring murmur, which terminates abruptly in the first sound. By combining palpation and auscultation the purring murmur is found to be synchronous with the thrill and the loud shock with the first sound. This is the presystolic murmur, about the time and mode of production of which so much discussion has occurred. I hold with those who regard it as occurring during the auricular systole. In whatever way produced, it remains one of the most distinctive and characteristic of murmurs and its presence is positively indicative of narrowing of the mitral orifice. The sole exception to this statement is the Flint murmur already referred to in aortic incompetency. Once, in a case of enormous enlargement of the

spleen, with dropsy, in which the heart was greatly pushed up, I heard a presystolic murmur of rough quality, and the mitral valves were found post mortem to be normal. The presystolic murmur may occupy the entire period of the diastole; but more commonly it is only the latter half, corresponding to the auricular systole. The difference may sometimes be noted between the first and second portions of the murmur, when it occupies the entire time. Often there is a peculiar rumbling or echoing quality, which in some instances is very limited and may be heard only over a single bell-space of the stethoscope. A systolic murmur may be heard at the apex or along the left sternal border, often of extreme softness and audible only when the breath is held. Sometimes the systolic murmur is loud and distinct and is transmitted to the axilla. The second sound in the second left interspace is loudly accentuated, sometimes reduplicated. It may be transmitted far to the left and be heard with great clearness beyond the apex. In uncomplicated cases of mitral stenosis there are usually no murmurs audible at the aortic region, at which spot the second sound is less intense than at the pulmonary area. In the lower sternum and to the right a tricuspid murmur is sometimes heard in advanced cases. Other points to be noted are the following: The unusually sharp, clear first sound which follows the presystolic murmur, the cause of which is by no means easy to explain. It can scarcely be a valvular sound produced chiefly at the mitral orifice, since it may be heard with great intensity in cases in which the valves are rigid and calcified. More probably it is a modified sound produced by the heart-muscle and connected, as has been suggested, with the altered conditions of the chordæ tendineæ and papillary muscles, the normal action of which must be interfered with.

These physical signs, it is to be borne in mind, are characteristic only of the stage in which compensation is maintained. Finally there comes a period in which, with rupture of compensation, the presystolic murmur disappears and there is heard in the apex region a sharp first sound, or sometimes a gallop rhythm. The marked systolic shock may be present after the disappearance of the thrill and the characteristic murmur. Under treatment, with gradual recovery of compensation, probably with increasing vigor of contraction of the right ventricle and left auricle, the presystolic murmur reappears. In cases seen at this stage of the disease the nature of the valve lesion may be entirely overlooked.

Stenosis of the mitral valve may for years be efficiently compensated by the hypertrophy of the right ventricle. Many persons with the characteristic physical signs of this lesion present no symptoms. They may for years perhaps be short of breath on going up-stairs, but are able to pass through the ordinary duties of life without discomfort. The pulse is smaller in volume than normal, but may be perfectly regular. A special danger of this stage is the recurring endocarditis. Vegetations may be whipped off into the circulation and, blocking a cerebral vessel, may cause hemiplegia or aphasia, or both. This, unfortunately, is not an uncommon

sequence in women. Patients with mitral stenosis may survive this accident for an indefinite period. A woman, over seventy years of age, died in one of my wards at the Philadelphia Hospital, who had been in the almshouse, hemiplegic, for more than thirty years. The heart presented an extreme grade of mitral stenosis which had probably existed at the time of the hemiplegic attack.

Failure of compensation brings in its train the group of symptoms which have been discussed under mitral insufficiency. Briefly enumerated they are: Rapid and irregular action of the heart, shortness of breath, cough, signs of pulmonary engorgement, and very frequently hæmoptysis. Attacks of this kind may recur for years. Bronchitis or a febrile attack may cause shortness of breath or slight blueness. Inflammatory affections of the lungs or pleura seriously disturb the right heart, and these patients stand pneumonia very badly. Many, perhaps a majority of cases of mitral stenosis, do not have dropsy. The liver may be greatly enlarged, and in the late stages ascites is not uncommon, particularly in children. General anasarca is most frequently met with in those cases in which there is secondary narrowing of the tricuspid orifice (Broadbent).

Tricuspid-Valve Disease.

(*a*) **Tricuspid Regurgitation.**—Occasionally this results from acute or chronic endocarditis with puckering; more commonly the condition is one of relative insufficiency, and is secondary to lesions of the valves on the left side, particularly of the mitral. It is met with also in all conditions of the lungs which cause obstruction to the circulation, such as cirrhosis and emphysema, particularly in combination with chronic bronchitis. The symptoms are those of obstruction in the lesser circulation with venous congestion in the systemic veins, such as has already been described in connection with mitral insufficiency. The signs of this condition are:

(1) Systolic regurgitation of the blood into the right auricle and the transmission of the pulse-wave into the veins of the neck. If the regurgitation is slight or the contraction of the ventricle is feeble there may be no venous pulsation, but in other cases there is marked systolic pulsation in the cervical veins. That in the right jugular is more forcible than that in the left. It may be seen both in the internal and the external, particularly in the latter. Marked pulsation in these veins occurs only when the valves guarding them become incompetent. Slight oscillations are by no means uncommon, even when the valves are intact. The distention of the veins is sometimes enormous, particularly in the act of coughing, when the right jugular at the root of the neck may stand out, forming an extraordinarily prominent ovoid mass. Occasionally the regurgitant pulse-wave may be widely transmitted and be seen in the subclavian and axillary veins, and even in the subcutaneous veins over the

shoulder, or, as in a case recently under observation, in the superficial mammary veins.

Regurgitant pulsation through the tricuspid orifice may be transmitted to the inferior cava, and so to the hepatic veins, causing a systolic distention of the liver. This is best appreciated by bimanual palpation, placing one hand over the fifth and sixth costal cartilages and the other in the lateral region of the liver in the mid-axillary line. The rhythmical expansile pulsation may be readily distinguished, as a rule, from the systolic depression of the liver due to communicated pulsation from the left ventricle.

(2) The second important symptom of tricuspid regurgitation is the occurrence of a systolic murmur of maximum intensity in the lower sternum. It is usually a soft, low murmur, often to be distinguished from a coexisting mitral murmur by differences in quality and pitch, and may be heard to the right as far as the axilla. Sometimes it is very limited in its distribution.

Together these two signs positively indicate tricuspid regurgitation. In addition, the percussion usually shows increase in the area of dulness to the right of the sternum, and the impulse in the lower sternal region is forcible. In the great majority of cases the symptoms are those of the associated lesions. In cirrhosis of the lung and in chronic emphysema the failure of compensation of the right ventricle with insufficiency of the tricuspid not infrequently leads either to acute asystole or to gradual failure with cardiac dropsy.

(*b*) **Tricuspid Stenosis.**—This interesting condition may be either congenital or acquired. The congenital cases are not uncommon, and are associated usually with other valvular defects which cause early death. The acquired form is not very infrequent. Bedford Fenwick collected 46 observations, of which 41 were in women. Leudet* has analyzed 117 cases. Of 101 of these in which the ages were mentioned, 80 were in women and 21 in men. A great majority of the cases were in adults, only eight being between the ages of ten and twenty. Its rarity as an isolated condition may be gathered from the fact that of 114 autopsies, in 11 only was the lesion confined to this valve. In 21 the tricuspid, mitral, and aortic segments were involved, and in 78 the tricuspid and mitral. Practically the condition is almost always secondary to lesions of the left heart.

The physical signs are sometimes characteristic. For instance, a presystolic thrill has been noted by several observers. The percussion shows dulness to be increased, particularly to the right of the sternum. On auscultation a presystolic murmur has been determined in certain cases, and is heard best at the root of the ensiform cartilage, or a little to the right of it. Of general symptoms, cyanosis of the face and lips is very common, and in the late stages, when dropsy supervenes, it is apt to be intense.

* Paris Thesis, 1888.

The lesion is interesting chiefly because it forms one of the most serious complications of mitral stenosis.

Pulmonary Valve Disease.

This is extremely rare.

(*a*) *Stenosis* is almost invariably a congenital anomaly. It constitutes one of the most important of the congenital cardiac affections. The valve segments are usually united, leaving a small, narrow orifice. In the adult cases occasionally occur. In Case 608 of my post-mortem records there was extreme stenosis in a girl of eighteen, owing to great thickening and adhesion of the segments, and there were also numerous vegetations. The orifice was only two millimetres in diameter. The congenital lesion is commonly associated with patency of the ductus Botalii and imperfection of the ventricular septum. There may also be tricuspid stenosis.

The physical signs are extremely uncertain. There may be a systolic murmur with a thrill heard best to the left of the sternum in the second intercostal space. This murmur may be very like a murmur of aortic stenosis, but is not transmitted into the vessels. Naturally the pulmonary second sound is weak or obliterated, or may be replaced by a diastolic murmur. Usually there is hypertrophy of the right heart.

(*b*) *Pulmonary Insufficiency.*—This rare affection is occasionally due to congenital malformation, particularly fusion of two of the segments. It is sometimes present, as Bramwell has shown, in cases of malignant endocarditis.

The physical signs are those of regurgitation into the right ventricle, but, as a rule, it is impossible to differentiate this from the murmur of aortic insufficiency, though the maximum intensity may be in the pulmonary area. In a recent case, in which two of the valve segments were closely glued to the wall of the pulmonary artery owing to the projection of an aneurism, a diastolic murmur developed under observation, which was transmitted loudly down the sternum. The condition is extremely rare and of little practical significance.

Combined Valvular Lesions.

These are extremely common. The mitral and aortic segments may be affected together; next in frequency comes the combination of mitral and tricuspid lesions; and then of aortic, mitral, and tricuspid. Aortic insufficiency or aortic stenosis is more frequently combined with mitral incompetency than aortic stenosis with mitral stenosis, or mitral stenosis with aortic insufficiency. In children the most common combination is aortic and mitral insufficiency. In adults, mitral insufficiency with thickening of the aortic valves and slight narrowing is perhaps the most common.

The diagnosis rests upon the character of the murmurs and the state of the chambers as regards hypertrophy and dilatation.

Prognosis in Valvular Disease.—The question is entirely one of efficient compensation. So long as this is maintained the patient may suffer no inconvenience, and even with the most serious forms of valve lesion the function of the heart may be little, if at all, disturbed.

Practitioners who are not adepts in auscultation and feel unable to estimate the value of the various heart murmurs should remember that the best judgment of the conditions may be gathered from inspection and palpation. With an apex beat in the normal situation and regular in rhythm the auscultatory phenomena may be practically disregarded.

As Sir Andrew Clark states, a murmur *per se* is of little or no moment in determining the prognosis in any given case. There is a large group of patients who present no other symptoms than a systolic murmur heard over the body of the heart, or over the apex, in whom the left ventricle is not hypertrophied, the heart rhythm is normal, and who may not have had rheumatism. Indeed, the condition is accidentally discovered, often during examination for life insurance. I know cases of this kind which have persisted unchanged for more than fifteen years Among the conditions influencing prognosis are:

(*a*) *Age.*—Children under ten are bad subjects. Compensation is well effected, and they are free from many of the influences which disturb compensation in adults. The coronary arteries also are healthy, and nutrition of the heart-muscle can be readily maintained. Yet, in spite of this, the outlook in cardiac lesions developing in very young children is usually bad. One reason is that the valve lesion itself is apt to be rapidly progressive, and the limit of cardiac reserve force is in such cases early reached. There seems to be proportionately a greater degree of hypertrophy and dilatation. Among other causes of the risks of this period are to be mentioned insufficient food in the poorer classes, the recurrence of rheumatic attacks, and the existence of pericardial adhesions The outlook in a child who can be carefully supervised and prevented from damaging himself by overexertion is naturally better than in one who is constantly overtasking his muscles. The valvular lesions which develop at, or subsequent to, the period of puberty are more likely to be permanently and efficiently compensated. Sudden death from heart-disease is very rare in children.

(*b*) *Sex.*—Women bear valve lesions, as a rule, better than men, owing partly to the fact that they live quieter lives, partly to the less common involvement of the coronary arteries, and to the greater frequency of mitral lesions. Pregnancy and parturition are disturbing factors, but are, I think, less serious than some writers would have us believe.

(*c*) *Valve affected.*—The relative prognosis of the different valve lesions is very difficult to estimate. Each case must, therefore, be judged on its own merits. Aortic insufficiency is unquestionably the most serious; yet

for years it may be perfectly compensated. Favorable circumstances in any case are the moderate grade of hypertrophy and dilatation, the absence of all symptoms of cardiac distress, and the absence of extensive arterio-sclerosis and of angina. The prognosis rests in reality with the condition of the coronary arteries. Rheumatic lesions of the valves, inducing insufficiency, are less apt to be associated with endarteritis at the root of the aorta; and in such cases the coronary arteries may escape for years. I know a physician, now about thirty-five years of age, who, when sixteen, had his first attack of rheumatism, which involved the aortic segments. He has had two subsequent attacks of rheumatism, but with care has been able to live a comfortable and fairly active life. On the other hand, when the aortic insufficiency is only a part of an extensive arterio-sclerosis at the root of the aorta, the coronary arteries are almost invariably involved, and the outlook in such cases is much more serious. Sudden death is not uncommon, either from acute dilatation during some exertion, or, more frequently, from blocking of one of the branches of the coronary arteries. The liability of this form to be associated with angina pectoris also adds to its severity. Aortic stenosis is a rare lesion, most commonly met with in middle-aged or elderly men, and is, as a rule, well compensated. In many cases it does not appear to limit the duration of life.

In mitral lesions the outlook on the whole is much more favorable than in aortic insufficiency. Mitral insufficiency, when well compensated, carries with it, perhaps, a better prognosis than mitral stenosis; but it must be borne in mind that the cases which last the longest are those in which the valve orifice is more or less narrowed, as well as incompetent. There is, in reality, no valve lesion so rapidly fatal and so poorly compensated as that in which the mitral segments are gradually curled and puckered until they form a narrow strip around a wide mitral ring—a condition specially seen in children. There are many cases of mitral insufficiency in which the defect is thoroughly balanced for thirty or even forty years, without distress or inconvenience. Even with great hypertrophy and the apex beat almost in the mid-axillary line, there may be little or no distress, and the compensation may be most effective. Women may pass safely through repeated pregnancies, though here they are liable to accidents associated with the severe strain. I have had under my care for many years a patient who had her first attack of rheumatism at the age of fifteen, when she already had a well-marked mitral murmur. When she first came under my observation, eighteen years ago, she had signs of hypertrophy of the left ventricle with a loud systolic murmur. She has had no cardiac disturbance whatever. She has lived a very active life, has been unusually vigorous, has borne eleven children, and has passed through three subsequent attacks of rheumatism.

In mitral stenosis the prognosis is usually regarded as less favorable. My own experience has led me, however, to place this lesion almost on a level, particularly in women, with the mitral insufficiency. It is found

very often in persons in perfect health, who have had neither palpitation nor signs of heart-failure, and who have lived laborious lives. The figures given, too, by Broadbent indicate that the date of death in mitral stenosis is comparatively advanced. These patients, too, pass through repeated pregnancies with safety. There are of course those too common accidents, the result of cerebral embolism, which are more liable to occur in this than in other forms.

Hard and fast lines cannot be drawn in the question of prognosis in valvular disease. Every case must be judged separately, and all the circumstances carefully balanced. There is no question which requires greater experience and more mature judgment, and even the most experienced are sometimes at fault.

The following brief summary of the conditions which justify a favorable prognosis embodies the large and varied clinical experience of Sir Andrew Clark: Good general health; just habits of living; no exceptional liability to rheumatic or catarrhal affections; origin of the valvular lesion independently of degeneration; existence of the valvular lesion without change for over three years; sound ventricles, of moderate frequency and general regularity of action; sound arteries, with a normal amount of blood and tension in the smaller vessels; free course of blood through the cervical veins; and, lastly, freedom from pulmonary, hepatic, and renal congestion.

Treatment of Valvular Lesions.—For this purpose the valvular lesion may be divided into the period of progressive development, with establishment and maintenance of hypertrophy, and the period of disturbed compensation.

(*a*) **Stage of Compensation.**—Medicinal treatment at this period is not necessary and is often hurtful. A very common error is to administer cardiac drugs, such as digitalis, on the discovery of a murmur or of hypertrophy. If the lesion has been found accidentally, it may be best not to tell the patient, but rather an intimate friend. Often it is necessary, however, to be perfectly frank in order that the patient may take certain preventive measures. He should lead a quiet, regulated, orderly life, free from excitement and worry. An ordinary wholesome diet should be taken, tobacco should be interdicted, and stimulants not allowed. Exercise should be regulated entirely by the feelings of the patient. So long as no cardiac distress or palpitation follows, moderate exercise will prove very beneficial. The skin should be kept active by a daily bath. Hot baths should be avoided and the Turkish bath should be interdicted. In the case of full-blooded, somewhat corpulent individuals an occasional saline purge should be taken. Patients with valvular lesions should not go into very high altitudes. The act of coition has serious risks, particularly in aortic insufficiency. Knowing that the causes which most surely and powerfully disturb the compensation are overexertion, mental worry, and malnutrition, the physician should give suitable instructions in each

case. As it is always better to have the co-operation of an intelligent patient, he should, as a rule, be told of the condition, but in this matter the physician must be guided by circumstances, and there are cases in which reticence is the wiser policy.

(*b*) **Stage of Broken Compensation.**—The break may be immediate and final, as when sudden death results from acute dilatation or from blocking of a branch of the coronary artery. Among the first indications are shortness of breath on exertion or attacks of nocturnal dyspnœa. These are often associated with impaired nutrition, particularly with anæmia, and a course of iron or change of air may suffice to relieve the symptoms.

Irregularity of the action of the heart cannot always be termed an indication of failing compensation, particularly in instances of mitral disease. It has greater significance in aortic lesions. Serious failure of compensation is indicated by signs of dilatation of the heart, the gallop rhythm, or various forms of arrhythmia, with or without the existence of dropsy. Under these circumstances the following measures are to be carried out:

(1) *Rest.*—Disturbed compensation may be completely restored by rest of the body. Both in Montreal and in Philadelphia it was a favorite demonstration in practical therapeutics to show the influence of complete rest and quiet on the cardiac dilatation. In many cases with œdema of the ankles, moderate dilatation of the heart, and irregularity of the pulse, the rest in bed, a few doses of the compound tincture of cardamoms, and a saline purge suffice, within a week or ten days, to restore the compensation. One patient, in Ward 11 of the Montreal General Hospital, with aortic insufficiency recovered from four successive attacks of failing compensation by these measures alone.

(2) The relief of the embarrassed circulation.

(*a*) *By Venesection.*—In cases of dilatation, from whatever cause, whether in mitral or aortic lesions or distention of the right ventricle in emphysema, when signs of venous engorgement are marked and when there is orthopnœa with cyanosis, the abstraction of from twenty to thirty ounces of blood is indicated. This is the occasion in which timely venesection may save the patient's life. It is a condition in which I have had most satisfactory results from venesection. It is done much better early than late. I have on several occasions regretted its postponement, particularly in instances of acute dilatation and cyanosis in connection with emphysema.*

(*b*) *By Depletion through the Bowels.*—This is particularly valuable when dropsy is present. Of the various purges the salines are to be preferred, and may be given by Matthew Hay's method. Half an hour to an hour before breakfast from half an ounce to an ounce and a half of Epsom salts may be given in a concentrated form. This usually produces

* For illustrative cases from my wards see paper by H. A. Lafleur, Medical News, July, 1891.

from three to five liquid evacuations. The compound jalap powder in half-drachm doses, or elaterium, may be employed for the same purpose. Even when the pulse is very feeble these hydragogue cathartics are well borne, and they deplete the portal system rapidly and efficiently.

(*c*) *The Use of Remedies which stimulate the Heart's Action.*—Of these, by far the most important is digitalis, which was introduced into practice by Withering. The indication for its use is dilatation; the contra-indication is a perfectly balanced compensatory hypertrophy, such as we see in all forms of valvular disease. Broken compensation, no matter what the valve lesion may be, is the signal for its use. It acts upon the heart, slowing and at the same time increasing the force of the pulsations. It acts on the peripheral arteries, raising their tension, so that a steady and equable flow of blood is maintained in the capillaries, which, after all, is the prime aim and object of the circulation. The beneficial effects are best seen in cases of mitral disease with small, irregular pulse and cardiac dropsy. Its effects are not less striking in the dilatation of the left ventricle, in the failing compensation of aortic insufficiency or of arterio-sclerosis. On theoretical grounds it has been urged that its use is not so advantageous in aortic insufficiency, since it prolongs the diastole and leads to greater distention. Practically, however, this need not be considered, and, when given with care, digitalis is just as serviceable in this as in any other condition associated with progressive dilatation. It may be given as the tincture or the infusion. In cases of cardiac dropsy, from whatever cause, fifteen minims of the tincture or half an ounce of the infusion may be given every three hours for two days, after which the dose may be reduced. Some prefer the tincture, others the infusion; it is a matter of indifference if the drug is good. The urine of a patient taking digitalis should be carefully estimated each day. As a rule, when its action is beneficial, there is within twenty-four hours an increase in the amount; often the flow is very great. Under its use the dyspnœa is relieved, the dropsy gradually disappears, the pulse becomes firmer, fuller in volume, and sometimes, if it has been very intermittent, regular.

Ill effects sometimes follow digitalis. There is no such thing as a cumulative action of the drug manifested by sudden symptoms. Toxic effects are seen in the production of nausea and vomiting. The pulse becomes irregular and small, and there may be two beats of the heart to one of the pulse, which, as pointed out by Broadbent, is found particularly in cases of mitral stenosis when they are under the influence of this drug. The urine is reduced in amount. These symptoms subside on the withdrawal of the digitalis, and are rarely serious. There are patients who take digitalis uninterruptedly for years, and feel palpitation and distress if the drug is omitted. In mitral disease, even when it does good it does not always steady the pulse. There are many cases in which the irregularity is not affected by the digitalis. When the compensation has been re-established the drug may be omitted. When there is dyspnœa on exer-

tion and cardiac distress, from five to ten minims three times a day may be advantageously given for protonged periods, but the effects should be carefully watched. In cardiac dropsy digitalis should be used at the outset with a free hand. Small doses should not be given, but from the first half-ounce doses of the infusion every three hours, or from fifteen to twenty minims of the tincture. There are no substitutes for digitalis.

Of other remedies strophanthus alone is of service. Given in doses of from five to eight minims of the tincture, it acts like digitalis. It certainly will sometimes steady the intermittent heart of mitral valve disease when digitalis fails to do so, but it is not to be compared with this drug when dropsy is present. Convallaria, citrate of caffeine, and *adonis vernalis* are warmly recommended as substitutes for digitalis, but their inferiority is so manifest that their use is rarely indicated.

There are two valuable adjuncts in the treatment of valvular disease—iron and strychnia. When anæmia is a marked feature iron should be given in full doses. In some instances of failing compensation iron is the only medicine needed to restore the balance. Arsenic is occasionally an excellent substitute, and one or other of them should be administered in all instances of heart-trouble when pallor is present. Strychnia is a heart tonic of very great value. It may be given in combination with the digitalis in one or two drop doses of the one per cent solution.

Treatment of Special Symptoms. (*a*) *Dropsy.*—The increased arterial tension and activity of the capillary circulation under the influence of digitalis hastens the interstitial lymph flow and favors resorption of the fluid. The hydragogue cathartics, by rapidly depleting the blood, promote the absorption of the fluid from the lymph spaces and the lymph sacs. These two measures usually suffice to rid the patient of the dropsy. In some cases, however, it cannot be relieved, and then Southey's tubes may be used or the legs punctured. If done with care, after a thorough washing of the parts, and if antiseptic precautions are taken, scarification is a very serviceable measure, and should be resorted to more frequently than it is. Canton-flannel bandages may be applied on the œdematous legs.

(*b*) *Dyspnœa.*—The patients are usually unable to lie down. A comfortable bed-rest should therefore be provided—if possible, one with lateral projections, so that in sleeping the head can be supported as it falls over. The shortness of breath is associated with dilatation, chronic bronchitis, or hydrothorax. The chest should be carefully examined in all these cases, as hydrothorax of one side or of both is a common cause of shortness of breath. There are cases of mitral regurgitation with recurring hydrothorax as the sole dropsical symptom, which is relieved, week by week or month by month, by tapping. For the nocturnal dyspnœa, particularly when combined with restlessness, morphia is invaluable and may be given without hesitation. The value of the calming influence of opium in all conditions of cardiac insufficiency is not enough recognized. There

are instances of cardiac dyspnœa unassociated with dropsy, particularly in mitral-valve disease, in which nitroglycerin is of great service, if given in the one per cent solution in increasing doses. It is especially serviceable in the cases in which the pulse tension is high.

(*c*) *Palpitation and Cardiac Distress.*—In instances of great hypertrophy and in the throbbing which is so distressing in some cases of aortic insufficiency, aconite is of service in doses of from one to three minims every two or three hours. An ice-bag over the heart or Leiter's coil is also of service in allaying the rapid action and the throbbing. For the pains, which are often so marked in aortic lesions, iodide of potassium in ten grain doses, three times a day, or the nitroglycerin may be tried. Small blisters are sometimes advantageous. It must be remembered that an important cause of palpitation and cardiac distress is flatulent distention of the colon, against which suitable measures must be directed.

(*d*) *Gastric Symptoms.*—The cases of cardiac insufficiency which do badly and fail to respond to digitalis are most often those in which nausea and vomiting are prominent features. The liver is often greatly enlarged in these cases; there is more or less stasis in the hepatic vessels, and but little can be expected of drugs until the venous engorgement is relieved. If the vomiting persists, it is best to stop the food and give small bits of ice, small quantities of milk and lime water, and effervescing drinks, such as Apollinaris water and champagne. Creosote, hydrocyanic acid, and the oxalate of cerium are sometimes useful; but, as a rule, the condition is obstinate and always serious.

(*e*) *Cough and Hæmoptysis.*—The former is almost a necessary concomitant of cardiac insufficiency, owing to engorgement of the vessels and more or less bronchitis. It is allayed by measures directed rather to the heart than to the lungs. Hæmoptysis in chronic valvular disease is sometimes a salutary symptom. An army surgeon, who was invalided during the late civil war on account of hæmoptysis, supposed to be due to tuberculosis, has since that time had, in association with mitral insufficiency and enlarged heart, many attacks of hæmoptysis. He assures me that his condition is invariably better after the attack. It is rarely fatal, except in some cases of acute dilatation, and seldom calls for special treatment.

(*f*) *Sleeplessness.*—One of the most distressing features of valvular lesions, even in the stage of compensation, is disturbed sleep. Patients may wake suddenly with throbbing of the heart, often in an attack of nightmare. Subsequently, when the compensation has failed, it is also a worrying symptom. The sleep is broken, restless, and frequently disturbed by frightful dreams. Sometimes a dose of the spirits of chloroform or of ether, with half a drachm of spirits of camphor, given in a little hot whisky, will give a quiet night. The compound spirits of ether, Hoffman's anodyne, though very unpleasant to take, is frequently a great boon in the intermediate period when compensation has partially failed

and the patients suffer from restless and sleepless nights. Paraldehyde and amylene hydrate are sometimes serviceable. Urethan, sulphonal, and chloralamide are rarely efficacious, and it is best, after a few trials, particularly if the paraldehyde does not answer, to give morphia. It may be given in combination with atropine.

(*g*) *Renal Symptoms.*—With ruptured compensation and lowering of the tension in the aorta, the urinary secretion is greatly diminished, and the amount may sink to five or six ounces in the day. Digitalis and strophanthus, when efficient, usually increase the flow. A brisk purge may be followed by augmented secretion. The combination in pill form of digitalis, squill, and the black oxide of mercury, will sometimes prove effective when the infusion or tincture of digitalis alone has failed. Calomel acts well in some cases, given in grs. iij every six hours for three or four days.

The *diet* in chronic valve diseases is often very difficult to regulate. With the dilatation and venous engorgement come nausea and often a great distaste for food. The amount of liquid should be restricted, and milk, beef-juice, or egg albumen given every three hours. When the serious symptoms have passed, eggs, scraped meat, fish, and fowl may be allowed. Starchy foods, and all articles likely to cause flatulency, should be forbidden. Stimulants are usually necessary, either whisky or brandy.

III. HYPERTROPHY AND DILATATION.

Hypertrophy is an enlargement of the heart due to an increased thickness, total or partial, in the muscular walls. Dilatation is an increase in size of one or more of the chambers with or without thickening of the walls. The conditions usually coexist, and could be more correctly described together under the term enlargement of the heart. Simple hypertrophy, in which the cavities remain of a normal size and the walls are increased, occurs, but simple dilatation, in which the cavities are increased and the walls remain of a normal diameter, probably does not, as it is always associated with thinning or with thickening of the coats. Commonly we have the forms of simple hypertrophy, hypertrophy with dilatation, and dilatation with thinning of the coats.

Hypertrophy of the Heart.

There are two forms—the simple hypertrophy, in which the cavity or cavities are of normal size; and hypertrophy with dilatation (eccentric hypertrophy), in which the cavities are enlarged and the walls increased in thickness. The condition formerly spoken of as concentric hypertrophy, in which there is diminution in the size of the cavity with thickening of the walls, is, as a rule, a post-mortem change.

The enlargement may affect the entire organ, one side, or only one chamber. Naturally, as the left ventricle does the chief work in forcing the blood through the systemic arteries, the change is most frequently found in it.

Etiology.—Hypertrophy of the heart follows the law governing muscles, that within certain limits, if the nutrition is kept up, increased work is followed by increased size—i. e., hypertrophy. Hypertrophy of the left ventricle alone, or with general enlargement of the heart, is brought about by—

Conditions affecting the heart itself: (1) Disease of the aortic valve; (2) mitral insufficiency; (3) general pericardial adhesions; (4) sclerotic myocarditis; (5) disturbed innervation, with overaction, as in exophthalmic goitre, in long-continued nervous palpitation, and as a result of the action of certain articles, such as tea, alcohol, and tobacco. In all of these conditions the work of the heart is increased. In the case of the valve lesions the increase is due to the increased intraventricular pressure; in the case of the adherent pericardium and myocarditis, to direct interference with the symmetrical and orderly contraction of the chambers.

Conditions acting upon the blood-vessels: (1) General arterio-sclerosis, with or without renal disease; (2) all states of increased arterial tension induced by the contraction of the smaller arteries under the influence of certain toxic substances, which act, as Bright suggested, by affecting "the minute capillary circulation, render greater action necessary to send the blood through the distant subdivisions of the vascular system"; (3) prolonged muscular exertion, which enormously increases the blood-pressure in the arteries; (4) narrowing of the aorta, as in the congenital stenosis.

Hypertrophy of the right ventricle is met with under the following conditions—

(1) Lesions of the mitral valve, either incompetence or stenosis, which act by increasing the resistance in the pulmonary vessels. (2) Pulmonary lesions, obliteration of any number of blood-vessels within the lungs, such as occurs in emphysema or cirrhosis, is followed by hypertrophy of the right ventricle. (3) Valvular lesions on the right side occasionally cause hypertrophy in the adult, not infrequently in the fœtus. (4) Chronic valvular disease of the left heart and pericardial adhesions are sooner or later associated with hypertrophy of the right ventricle.

In the auricles simple hypertrophy is never seen; it is always dilatation with hypertrophy. In the left auricle the condition develops in lesions at the mitral orifice, particularly stenosis. The right auricle hypertrophies when there is greatly increased blood-pressure in the lesser circulation, whether due to mitral stenosis or pulmonary lesions. Narrowing of the tricuspid orifice is a less frequent cause.

Morbid Anatomy.—The heart of an average-sized man weighs about nine ounces (280 grammes); that of a woman, about eight ounces (250 grammes). In cases of general hypertrophy the heart may weigh

from sixteen to twenty ounces. Weights above twenty-five ounces are rare. So far as I know, the heaviest heart on record is one described by Beverly Robinson, weighing fifty-three ounces. Dulles has reported one weighing forty-eight ounces. The measurement of the thickness of the walls is, next to weighing, the best means of determining the hypertrophy. In extreme dilatation the walls, though actually thickened, may look thin. When *rigor mortis* is present, the cavity may be small and the walls may appear greatly thickened. The measurements should not be made until the heart has been soaked in water and thoroughly relaxed. In the left ventricle a thickness of ten lines, or from twenty to twenty-five millimetres, indicates hypertrophy. The right ventricle is thinner than the left, and has an average diameter of from four to seven millimetres. In hypertrophy it may measure from thirteen to twenty millimetres. The left auricle has a normal thickness of about three millimetres, which may be doubled in hypertrophy. The wall of the right auricle is thinner than that of the left, rarely exceeding two millimetres in diameter. The appendices of the auricles often present marked increase in thickness and the musculi pectinati are greatly developed.

The shape of the heart is altered in hypertrophy; with great enlargement of the ventricles, the apex is broadened, and the conical shape is lost. In the enormous enlargement of aortic insufficiency this rotundity of the apex is very marked. When the right ventricle is chiefly affected it occupies the largest share of the apex. In mitral stenosis the contrast is very striking between the large, broad right ventricle, reaching to the apex, and the small left chamber.

The hypertrophied muscle has a deep red color, is firm, and is cut with increasing resistance. The right ventricle, as Rokitansky noted, may have a peculiar hard, leathery consistence. In simple hypertrophy of the left ventricle the papillary muscles and the columnæ carneæ may be enlarged, but the former are often much flattened in dilated hypertrophy. The muscular trabeculæ are more developed, as a rule, in the right ventricle than in the left.

The increase in size of the heart is probably due to a definite numerical increase, resulting from development of new fibres.

Symptoms.—Hypertrophy is a conservative process, secondary to some valvular or arterial lesion, and is not necessarily accompanied by symptoms. So admirable is the adjusting power of the heart that, for example, an advancing stenosis of aortic or mitral orifice may for years be perfectly equalized by a progressive hypertrophy, and the subject of the affection be happily unconscious of the existence of heart-trouble. Hypertrophy is in almost all cases an unmixed good; the symptoms which arise are usually to be attributed to its failure, or, as we say, to disturbance of compensation.

Among the most common symptoms are unpleasant feelings about the heart—a sense of fulness and discomfort, rarely amounting to pain. This

may be very noticeable when the patient is recumbent on the left side. Actual pain is rare, except in the irritable heart from tobacco or in neurasthenics. Palpitation may not occur, nor do patients always have sensations from the violent shocks of a greatly hypertrophied organ. There are instances in which very uneasy feelings arise from a moderately exaggerated pulsation. The general condition has much to do with this. In health we are not conscious of the heart's pulsations, but one of the first indications of exhaustion from excesses or overstudy is the consciousness of the heart's action, not necessarily with palpitation. Headaches, flushings of the face, noises in the ears, and flashes of light may be present.

Certain untoward effects of long-continued hypertrophy of the left ventricle must be mentioned, chief among which is the production of arterio-sclerosis. Particularly is this the case when the hypertrophy results from increased peripheral resistance. The heightened blood-pressure (expressed by the word strain) in the arteries gradually induces an endarteritis and a stiff, inelastic state of those vessels most exposed to it—viz., the aorta and its primary divisions. In overcoming the peripheral obstruction the hypertrophy "ruins the arteries as a sequential result" (Fothergill). Prolonged muscular exertion also acts injuriously in this way.

Another danger is rupture of the blood-vessels, particularly those of the brain. In general arterial degeneration associated with contracted kidneys and hypertrophied left heart apoplexy is common. Indeed, in the majority of cases of cerebral hæmorrhage there is sclerosis of the smaller vessels, often with the development of miliary aneurisms, and the rupture may be caused by the forcible action of the heart.

Physical Signs.—*Inspection* may show bulging of the præcordia, producing in children marked asymmetry of the chest. It may occur without pericardial adhesions, which Schroetter thinks are invariably associated with this condition. The intercostal spaces are widened, and the area of visible impulse is much increased. On *palpation* the impulse is forcible and heaving, and with each systole the hand or the ear applied over the heart may be visibly raised. A slow, heaving impulse is one of the best signs of simple hypertrophy. With large dilated hypertrophy the forcible impulse is often more sudden and abrupt. A second, weaker impulse can sometimes be felt, due perhaps to a rebound from the aortic valves (Gowers). The beat may be felt in the sixth, seventh, or eighth interspace from one to three inches outside the nipple. This downward dislocation of the apex is an important sign in hypertrophy of the left ventricle. In moderate grades, such as are seen in chronic Bright's disease, the impulse may be in the sixth interspace in the nipple line, or a little outside of it.

Percussion reveals increased dulness, which in the parasternal line may begin at the third rib or in the second interspace, and transversely may extend from half an inch to two inches beyond the nipple line and an equal distance beyond the middle line of the sternum. The dull area

is more ovoid than in health. When carefully delimited the colossal hypertrophy of aortic valve disease may give an area of dulness from seven to eight inches in transverse extent. In moderate grades a transverse dulness of four inches is not uncommon.

On *auscultation* the sounds, when the valves are healthy, may present no special changes, but the first sound is often prolonged and dull. When there is dilatation as well, it may be very clear and sharp. Reduplication is common in the hypertrophy of renal disease. A peculiar clink —the *tintement métallique* of Bouillaud—may be heard just to the right of the apex beat. The second sound is clear and loud, sometimes ringing in character or reduplicated. With valvular lesions, the sounds, of course, are much altered, and are replaced or accompanied by murmurs.

In simple hypertrophy not dependent on valvular lesions, the pulse is usually regular, full, strong, and of high tension. It may be increased in rapidity, but is often normal. In eccentric hypertrophy the pulse is full, but softer, and usually more rapid. One of the earliest signs of failure and dilatation is irregularity and intermittence of the pulse.

Hypertrophy of the *right ventricle* in the adult very rarely follows valvular disease on the right side, but results from increased resistance in the pulmonary circulation, as in cirrhosis of the lung and emphysema, or in stenosis of the mitral orifice. With perfect compensation, which fully maintains the equilibrium of the circulation, there are no symptoms. Extra exertion, as the ascent of stairs or running, may cause shortness of breath, but in many ways hypertrophy of the right ventricle is the most enduring and salutary form in the whole cycle of cardiac affections. For long periods of years the effects of mitral stenosis may be counterbalanced, and only sudden death by accident or an acute disease reveal the existence of an unsuspected lesion. In the hypertrophy secondary to emphysema or cirrhosis of the lungs, there may be sensations of distress in the cardiac region, with cough and shortness of breath; but as long as the dilatation is moderate the symptoms are not marked. With great dilatation and tricuspid leakage come venous engorgement, œdema, and pulmonary troubles. The increased pressure in the lesser circulation leads to sclerosis of the pulmonary arteries and the constant engorgement of the capillaries leads ultimately to a deposition of pigment and increase in the fibrous elements in the lung—the brown induration. Extreme pulmonary congestion and apoplexy are more often associated with dilatation. Hæmoptysis may result from rupture of vessels during sudden exertion.

Physical Signs.—Bulging of the lower part of the sternum and left cartilages occurs. The apex beat is forced to the left, but is not so often displaced downward. The most marked impulse may be in the angle between the ensiform cartilage and the seventh rib or beneath the cartilages of the sixth and seventh ribs. The pulsation is rather diffuse, not punctuate, particularly if there is much dilatation. In thin-walled chests there may be pulsation in the third and fourth right interspaces.

The cardiac dulness is increased transversely and toward the right; it may extend an inch or more beyond the border of the sternum. On auscultation the first sound at the lower part of the sternum is louder and fuller than normal, but the differences are not very marked unless there is much dilatation, when the sound is clearer and sharper. Accentuation and reduplication of the second sound are heard in the pulmonary artery on account of the increased tension. The pulse at the wrist is usually small. Pulsation occurs in the jugulars when there is tricuspid incompetence.

Hypertrophy of the *auricles* always occurs with dilatation. It is most common in the left chamber, which hypertrophies in mitral stenosis and incompetency and naturally assists in restoring the balance of the circulation. There are no distinctive physical signs, and we usually can infer its presence only by the existence of mitral stenosis and a presystolic murmur. Increased dulness may be determined to the left of the sternum, and there may be a presystolic wave in the second left interspace.

Hypertrophy and dilatation of the right auricle are met with (associated with a similar condition in the right ventricle and incompetency of the tricuspid) in emphysema, cirrhosis of the lung, chronic bronchitis, and mitral disease. In comparison with the left auricle the greater development and hypertrophy of the appendix and its musculi pectinati is very striking. The latter may be distributed over the anterior wall of the sinus to a greater extent than in health. There are increased dulness in the third and fourth interspaces, pulsation sometimes presystolic in rhythm, signs of venous engorgement, jugular pulsation, and other evidences of dilatation of the right heart.

Diagnosis.—Among conditions to be distinguished are:

(1) Neurotic palpitation, from whatever cause, even when very forcible, has not the heaving impulse of genuine hypertrophy. Enlargement of the organ may, however, follow prolonged overaction, as in the smoker's heart, the irritable heart of neurasthenics, and in exophthalmic goitre, but it is usually slight.

(2) The increased area of dulness may be due to a variety of causes, some of which may closely simulate hypertrophy, such as pericardial effusion, aneurism, mediastinal growths, or displacement of the heart from pressure, or the existence of malformation of the chest. With the exercise of ordinary care, however, the diagnosis can usually be made. There are two opposite conditions which frequently give trouble. With the left lung contracted from pleurisy, phthisis, or cirrhosis, a large surface of the heart is exposed; the pulsation may be extensive and forcible, and may at first sight resemble hypertrophy. In this condition there is dislocation upward and to the left. The existence of pulmonary or pleuritic disease and the fixation of the lung on deep inspiration will suffice to prevent mistakes. A less extensive exposure of the heart may occur without any disease in very narrow-chested persons with ill-developed lungs; here, though the area of dulness may be much increased, the normal position

of the apex, the absence of forcible, heaving impulse, and of any obvious cause of hypertrophy will afford satisfactory criteria for a diagnosis. The reverse condition exists in some cases in which emphysema masks moderate cardiac hypertrophy. The area of dulness may be normal, or even diminished, and the pulse and character of the sounds will help in the diagnosis; but it is sometimes a difficult matter.

Prognosis.—The course of any case of cardiac hypertrophy may be divided into three stages:

(*a*) The period of development, which varies with the nature of the primary lesion. For example, in rupture of an aortic valve, during a sudden exertion, it may require months before the hypertrophy becomes fully developed; or, indeed, it may never do so, and death may follow from an uncompensated dilatation. On the other hand, in sclerotic affections of the valves, with stenosis or incompetency, the hypertrophy develops step by step with the lesion, and may continue to counterbalance the progressive and increasing impairment of the valve.

(*b*) The period of full compensation—the latent stage—during which the heart's vigor meets the requirements of the circulation. This period may last an indefinite time, and a patient may never be made aware by any symptoms that he has a valvular lesion.

(*c*) The period of broken compensation, which may come on suddenly during very severe exertion. Death may result from acute dilatation; but more commonly it takes place slowly and results from degeneration and weakening of the heart-muscle.

The breaking or rupture of cardiac compensation may be induced by many causes, among which the most important are: (1) Failure of the general nutrition. In many instances of heart-disease, exposure, poor food, and alcohol combine to bring about disturbance of a well-balanced heart lesion. Acute illnesses, particularly the fevers, may induce general debility and with it weakening of the heart-muscle. (2) Disturbance of the local nutrition of the heart, owing to gradual sclerosis of the coronary arteries, is a common cause. (3) Very severe muscular exertion, which may disturb a compensation, perfect for years, and induce death in a few days (Traube). (4) Mental emotions. Severe grief or fright may bring on failure of compensation.

The prognosis is largely, as already stated, a matter of maintained compensation. Once established, the hypertrophy rarely, if ever, disappears, inasmuch as the cause usually persists. Occasionally, perhaps, the hypertrophy associated with neurotic palpitation from tobacco, or other causes, or the hypertrophy following muscular over-exertion, may disappear.

Dilatation of the Heart.

Two varieties are recognized, dilatation with thickening and dilatation with thinning. The former is the most common, and corresponds to the dilated or eccentric hypertrophy.

Etiology.—Two important causes combine to produce dilatation—increased pressure within the cavities and impaired resistance, due to weakening of the muscular wall—which may act singly, but are often combined. A weakened wall may yield to a normal distending force, or a normal wall may yield under a heightened blood-pressure.

(1) Heightened endocardiac pressure results either from an increased quantity of blood to be moved or an obstacle to be overcome, and is the most frequent cause. It does not necessarily bring about dilatation; simple hypertrophy may follow, as in the early period of aortic stenosis, and in the hypertrophy of the left ventricle in Bright's disease.

A majority of the important causes of increased endocardiac pressure have already been discussed under hypertrophy. One or two may be considered more in detail.

The size of the cardiac chambers varies in health. With slow action of the heart the dilatation is complete and fuller than it is with rapid action. Physiologically, the limits of dilatation are reached when the chamber does not empty itself during the systole. This may occur as an acute, transient condition in severe exertion—during, for example, the ascent of a mountain. There may be great dilatation of the right heart, as shown by the increased epigastric pulsation, and even increase in the cardiac dulness. The safety-valve action of the tricuspid valves may here come into play, relieving the lungs by permitting regurgitation into the auricle. With rest the condition is removed, but if it has been extreme, the heart may suffer a strain from which it may recover slowly, or, indeed, the individual may never be able again to undertake severe exertion. In the process of training, the getting wind, as it is called, is largely a gradual increase in the capability of the heart, particularly of the right chambers. A degree of exertion can be safely maintained in full training which would be quite impossible under other circumstances, because by a gradual process of what we may call physical education the heart has strengthened its reserve force—widened enormously its limits of physiological work. Endurance in prolonged contests is measured by the capabilities of the heart, and its essence consists in being able to meet the continuous tendency to overstep the limits of dilatation.

We have no positive knowledge of the nature of the changes in the heart which occur in this process, but it must be in the direction of increased muscular and nervous energy. The large heart of athletes may be due to the prolonged use of their muscles, but no man becomes a great runner or oarsman who has not naturally a capable if not a large heart. Master McGrath, the celebrated greyhound, and Eclipse, the

race-horse, both famous for endurance rather than speed, had very large hearts.

Excessive dilatation during severe muscular effort results in heart-strain. A man, perhaps in poor condition, calls upon his heart for extra work during the ascent of a high mountain, and is at once seized with pain about the heart and a sense of distress in the epigastrium. He breathes rapidly for some time, is "puffed," as we say, but the symptoms pass off after a night's quiet. An attempt to repeat the exercise is followed by another attack, or, indeed, an attack of cardiac dyspnœa may come on while he is at rest. For months such a man may be unfitted for severe exertion, or he may be permanently incapacitated. In some way he has overstrained his heart and become "broken-winded." Exactly what has taken place in these hearts we cannot say, but their reserve force is lost, and with it the power of meeting the demands exacted in maintaining the circulation during severe exertion. The "heart-shock" of Latham includes cases of this nature—sudden cardiac breakdown during exertion, not due to rupture of a valve. It seems probable that sudden death in men during long-continued efforts, as in a race, is sometimes due to over-distention and paralysis of the heart.

Examples of dilatation occur in all forms of valve lesions. In aortic incompetency blood enters the left ventricle during diastole from the unguarded aorta and from the left auricle, and the quantity of blood at the termination of diastole subjects the walls to an extreme degree of pressure, under which they inevitably yield. In time they augment in thickness, and present the typical eccentric hypertrophy of this condition.

In mitral insufficiency blood which should have been driven into the aorta is forced into and dilates the auricle from which it came, and then in the diastole of the ventricle a large amount is returned from the auricle, and with increased force. In mitral stenosis the left auricle is the seat of greatly increased tension during diastole, and dilates as well as hypertrophies; the distention, too, may be enormous. Dilatation of the right ventricle is produced by a number of conditions, which were considered under hypertrophy. All circumstances, such as mitral stenosis, emphysema, etc., which permanently increase the tension of the blood in the pulmonary vessels, will cause its dilatation.

(2) Impaired nutrition of the heart-walls may lead to a diminution of the resisting power so that dilatation readily occurs.

The loss of tone due to parenchymatous degeneration or myocarditis in fevers may lead to a fatal condition of acute dilatation. It is a recognized cause of death in scarlatinal dropsy (Goodhart), and may occur in rheumatic fever, typhus, typhoid, erysipelas, etc. The changes in the heart-muscle which accompany acute endocarditis or pericarditis may lead to dilatation, especially in the latter disease. In anæmia, leukæmia, and chlorosis the dilatation may be considerable. In sclerosis of the walls, the yielding is always where this process is most advanced, as at the left apex.

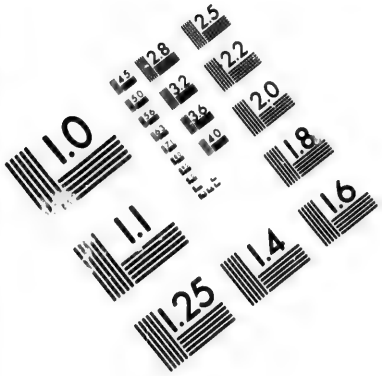
Under any of these circumstances the walls may yield with normal blood-pressure.

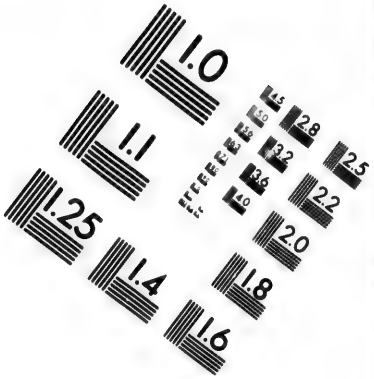
Pericardial adhesions are a cause of dilatation, and we generally find in cases with extensive and firm union considerable hypertrophy and dilatation. There is usually here some impairment as well of the superficial layers of muscle.

Morbid Anatomy.—The condition usually exists with hypertrophy in two or more chambers. It is more common on the right than on the left side. The most extreme dilatation is in cases of aortic incompetency, in which all the cavities may be enormously distended. In mitral stenosis the left auricle is often trebled in capacity, and the right chambers also are very capacious. The auricles may contain from eighteen to twenty ounces of blood. In chronic lesions of the lungs the right chambers are chiefly involved. In great distention of one ventricle the septum may bulge toward the other side. The auriculo-ventricular rings are often dilated, and there may be an increase in the circumference of an inch and a half or even two inches. Thus, the tricuspid orifice, the circumference of which is about four and a half inches, may freely admit a graduated heart-cone of over six inches; and the mitral orifice, which normally is about three and a half inches, may admit the cone to five and a half inches or even more. Great dilatation is always accompanied with relative incompetency of the valves, so that free regurgitation into the auricles is permitted. The orifices of the venæ cavæ and of the pulmonary veins may be greatly dilated.

The endocardium is often opaque, particularly that of the auricles. The muscle substance varies according to the presence or absence of degenerations. The microscope may show marked fatty or parenchymatous change, but in some instances no special alteration may be noticeable. There is much truth in Niemeyer's assertion "that it is not possible by means of the microscope to recognize all the alterations of the muscular fibrillæ which diminish the functional power of the heart." Of the changes in the ganglia of the heart we know very little. As centres of control they probably have more to do with cardiac atony and breakdown than we generally admit. Degeneration of them has been noted by Putjakin, Ott, and others.

Symptoms and Physical Signs.—Dilatation causes weakness of the cardiac walls, diminishes the vigor of their contractions, and is therefore the reverse of hypertrophy. So long as compensation is maintained the enlargement of a cavity may be considerable. The limit is reached when the hypertrophied walls in the systole can no longer expel all the contents, part of which remain, so that at each diastole the chamber is abnormally full. Thus, in aortic incompetency blood enters the left ventricle from the aorta as well as the auricle; dilatation ensues, and also hypertrophy as a direct effect of the increased pressure and increased amount of blood to be moved. But if from any cause the hypertrophy





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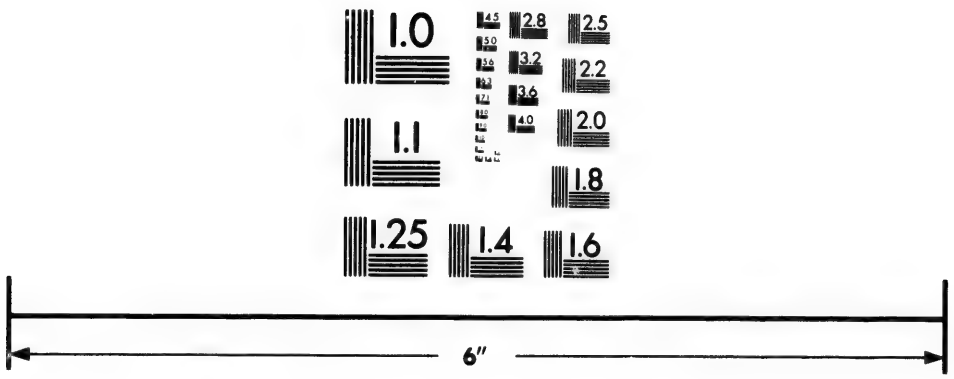

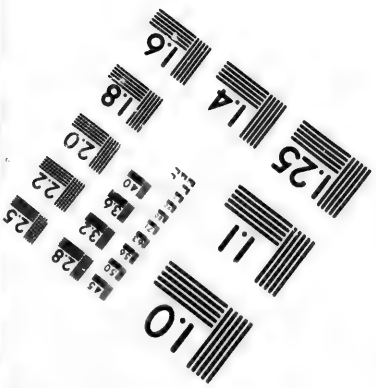

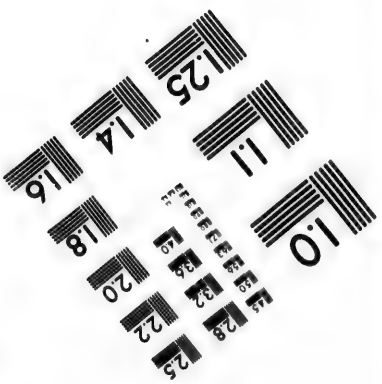

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weakens and the ventricle during systole fails to empty itself completely, a still larger amount is in it at the end of each diastole, and the dilatation becomes greater. The amount remaining after systole prevents the blood from entering freely from the auricle. Incompetency of the auriculo-ventricular valves follows, with dilatation of the auricle and impeded blood-flow in the pulmonary veins. Dilatation and hypertrophy of the right heart may compensate for a time, but when this fails the venous system becomes engorged and dropsy may result. The consideration of the symptoms of chronic valvular lesions is largely that of dilatation and its effects. Acute dilatation, such as we see in fevers or in sudden failure of a hypertrophied heart, is accompanied by three chief symptoms—weak, usually rapid, impulse, dyspnœa, and signs of obstructed venous circulation. Cardiac pain may be present, but is often absent.

The *physical signs* of dilatation are those of a weak and enlarged organ. The impulse is diffuse, often undulatory, and is felt over a wide area, and an apex beat or a point of maximum intensity may not exist. When it does exist, it may be visible and yet cannot be felt—a valuable observation made by Walshe. An extensive area of impulse with a quick, weak maximum apex beat may be present. When the right heart is chiefly dilated the left may be pushed over so as to occupy a much less extensive area in front of the heart, and the true apex beat cannot be felt; but the chief impulse is just below, or to the right of, the xiphoid cartilage, and there is a wavy pulsation in the fourth, fifth, and sixth interspaces to the left of the sternum. In extreme dilatation of the right auricle a pulsation may sometimes be seen in the third right interspace close to the sternum, and with free tricuspid regurgitation this may be systolic in character. Whether the pulsation frequently seen in the second left interspace is ever due to a dilated left auricle has not been determined. I have sometimes thought it was presystolic in rhythm, though it may be distinctly systolic. Post mortem, it is rare in the most extreme distention to see the auricular appendix so far forward as to warrant the belief that it could beat against the second interspace. The area of dulness is increased, but an emphysematous lung or the fully distended organ in a state of brown induration may cover over the heart and greatly limit the extent. The directions of increase were considered in connection with hypertrophy.

The first sound is shorter, sharper, more valvular in character, and more like the second. As the dilatation becomes excessive it gets weaker. Reduplication is not common, but occasionally differences may be heard in the first sound over the right and left hearts. The sounds are frequently obscured by murmurs, which are produced by incompetency of the valves due to the great dilatation, or are associated with the chronic valve disease on which the condition depends. The aortic second sound is replaced by a murmur in aortic regurgitation. The pulmonary sound is accentuated in mitral regurgitation and pulmonary congestion, but

with extreme dilatation it may be much weakened. The heart's action is irregular and intermittent, and the pulse is small, weak, and quick.

On auscultation both the sounds may be free from murmur. Often there is the condition known as embryocardia or fœtal heart-rhythm, in which the first and second sounds are very alike, and the long pause is shortened. In other instances there is the typical and characteristic gallop rhythm, rarely found apart from conditions of dilatation. With the various valvular lesions the corresponding murmurs may be heard. Murmurs, however, which have been present may disappear, as in the case of mitral stenosis. In other instances a loud systolic murmur may be heard at the apex, and when the case first comes under observation it may be impossible to say whether this is due to organic mitral lesion. The murmur may be confined to the apex region, or propagated well to the back. It is extremely common in the dilatation which follows the hypertrophy of the left ventricle in arterio-sclerosis. Under treatment, with the gradual disappearance of the dilatation, a murmur of this kind, even though most intense, may completely disappear, showing that it has been due to a relative insufficiency, not to a valvular lesion. All varieties of arrhythmia may occur in dilatation of the heart. The pulse, as a rule, is small, weak, quick, and often irregular.

Dilatation and Hypertrophy due to Overexertion and Alcohol.—There is a group of cases of dilatation and hypertrophy dependent upon prolonged overexertion, which rarely comes under observation until compensation has failed, and which then may be very difficult to distinguish from the similar conditions produced by valvular disease. The patients are able-bodied men at the middle period of life, and complain first of palpitation or irregularity of the action of the heart, shortness of breath, and subsequently the usual symptoms of cardiac insufficiency develop. On inquiring into the history of these patients none of the usual etiological factors causing valve disease are present, but they have always been engaged in laborious occupations and have usually been in the habit of taking stimulants freely. This is the affection which has been specially studied by McLean, Clifford Albutt, Seitz, and others, and in its earlier condition by Da Costa, in what he termed the irritable heart It is met with very frequently in soldiers. These cases may return to hospital three or four times with cardiac insufficiency, sometimes with slight anasarca, hæmoptysis, and signs of pulmonary engorgement. The condition is by no means infrequent. Bollinger has called attention to the common occurrence of dilatation and hypertrophy in beer-drinkers, particularly in the workers in the German breweries, who drink twenty or more litres in the day. Strümpell, at his Erlangen clinic, told me that this condition was very common in the draymen and workers in the breweries of that town, very few of whom pass the forty-fifth year without indications of hypertrophy and dilatation of the heart. On post-mortem examination the valves may be quite healthy, the aorta smooth, and no extensive arterio-

sclerosis or renal disease. The heart weighs from eighteen to twenty-five ounces; the chambers are dilated. The condition has been met with also in animals, and Houghton states that the heart of the celebrated greyhound Master McGrath weighed 9·57 ounces, just threefold in excess of the normal proportion of heart-weight to body-weight.

Idiopathic Dilatation.—And, lastly, there are other cases in which dilatation of the heart occurs without discoverable cause. In some instances there has been a history of sudden exercise or of mental emotion, but in other cases the condition seems to have come on spontaneously. In some the condition is acute and the patient has dyspnœa, slight cyanosis, cough, and great cardiac distress. Death may occur in a few days, or dropsy may supervene and the case may become chronic. Delafield has reported an interesting series of cases of this group.

Treatment.—The treatment of hypertrophy and dilatation has already been considered under the section on valvular lesions. I would only here emphasize the fact that with signs of dilatation, as indicated by gallop rhythm, urgent dyspnœa, and slight lividity, venesection is in many cases the only means by which the life of the patient may be saved, and from twenty-five to thirty ounces of blood should be abstracted without delay. Subsequently stimulants, such as ammonia and digitalis, may be administered, but they are accessories only to the bleeding in the critical condition of acute dilatation, which is so frequently met with in cardiac lesions.

IV. AFFECTIONS OF THE MYOCARDIUM.

1. **Lesions due to Disease of the Coronary Arteries.**—A knowledge of the changes produced in the myocardium by disease of the coronary vessels gives a key to the understanding of many problems in cardiac pathology. The terminal branches of the coronary vessels are end arteries. The blocking of one of these vessels by a thrombus or an embolus leads to a condition which is known as—

(*a*) *Anæmic necrosis*, or white infarct. This is most commonly seen in the left ventricle and in the septum, in the territory of distribution of the anterior coronary artery. The affected area has a yellowish-white color, sometimes a turbid, parboiled aspect, at others a grayish-red tint. It may be somewhat wedge-shaped, more often it is irregular in contour and projects above the surface. Microscopically the changes are very characteristic. The nuclei disappear from the muscle fibres, the condition of fragmentation is present, and the fibres present a homogeneous, hyaline appearance. In some instances there is complete transformation, and even to the naked eye a firm white patch of hyaline degeneration may appear in the centre of the area. Sudden death not infrequently follows the blocking of one of the branches of the coronary

artery and the production of this anæmic necrosis. *In medico-legal cases it is a point of primary importance to remember that this is one of the common causes of sudden death.* This condition should be carefully sought for, inasmuch as it may be the sole lesion, except a general, sometimes slight arterio-sclerosis. Rupture of the heart may be associated with anæmic necrosis.

(*b*) The second important effect of coronary-artery disease upon the myocardium is seen in the production of *fibrous myocarditis.* This may result from the gradual transformation of areas of anæmic necrosis. More commonly it is caused by the narrowing of a coronary branch in a process of obliterative endarteritis. The sclerosis is most frequently seen at the apex of the left ventricle and in the septum, but it may occur in any portion. In the septum often there are streaks of fibroid degeneration which do not reach the endocardium, and it may be necessary to divide the muscle in order to see them. Hypertrophy of the heart is commonly associated with this degeneration. It is the invariable precursor of aneurism of the heart.

Complete obliteration of one coronary artery, if produced suddenly, is usually fatal. When induced slowly, either by arterio-sclerosis at the orifice of the artery at the root of the aorta or by an obliterating endarteritis in the course of the vessel, the circulation may be carried on through the other vessel. Sudden death is not uncommon, owing to thrombosis of a vessel which has become narrowed by sclerosis. In the most extreme grade one coronary artery may be entirely blocked, with the production of extensive fibroid disease, and a main branch of the other also may be occluded. A large, powerfully built imbecile, aged thirty-five, at the Elwyn Institution, Pennsylvania, who had for years enjoyed doing the heavy work about the place, died suddenly, without any preliminary symptoms. The heart, which is in my collection, weighed over twenty ounces; the anterior coronary artery was practically occluded by obliterating endarteritis, and of the posterior artery one main branch was occluded.

(*c*) *Septic Infarcts.*—In pyæmia the smaller branches of the coronary arteries may be blocked with septic emboli and cause infarcts in the myocardium in the form of miliary abscesses, varying in size from a pea to a pin's head. These may not cause any disturbance, but when large they may perforate into the ventricle or into the pericardium, forming what has been called acute ulcer of the heart.

2. **Acute Interstitial Myocarditis.**—In the fevers and in pericarditis the intermuscular connective tissue is swollen and infiltrated with round cells and nuclei, the vessels are dilated, there are minute extravasations, and the muscle fibres may be granular or fatty, with indistinct striæ and nuclei. These instances have been met with in typhoid fever, smallpox, and diphtheria. The muscle substance is pale, soft, and easily torn, and the condition has been described either as inflammatory or degenerative.

3. **Parenchymatous Degeneration.**—This is usually met with in fevers, or in connection with endocarditis or pericarditis. It is characterized by a pale, turbid state of the cardiac muscle, which is general, not localized. Turbidity and softness are the special features. It is the softened heart of Laennec and Louis. Stokes speaks of an instance in which "so great was the softening of the organ that when the heart was grasped by the great vessels and held with the apex pointing upward, it fell down over the hand, covering it like a cap of a large mushroom."

Histologically, there is a degeneration of the muscle fibres, which are infiltrated to a various extent with granules which resist the action of ether. Sometimes this granular change in the fibres is extreme, and no trace of the striæ can be detected. It is probably the effect of a toxic agent, and is seen in its most exquisite form in the lumbar muscles in cases of toxic hæmoglobinuria in the horse. It is met with in cases of typhoid, typhus, small-pox, and other infectious diseases, particularly when the course is protracted. There is no definite relation between it and the high temperature.

A form of myocarditis has been described, characterized by fragmentation of the fibres owing to softening of the cement substance. According to von Recklinghausen this is a post-mortem change.

4. **Fatty Heart.**—Under this term are embraced fatty degeneration and fatty overgrowth.

(*a*) *Fatty degeneration* is a very common condition, and mild grades are met with in many diseases. It is found in the failing nutrition of old age, of wasting diseases, and of cachectic states; in prolonged infectious fevers, in which it may follow or accompany the parenchymatous change; associated with acute and chronic anæmias. Certain poisons, such as phosphorus, produce an intense fatty degeneration. Local causes: Pericarditis is usually associated with fatty or parenchymatous changes in the superficial layers of the myocardium. Disease of the coronary arteries is a common and important cause. Lastly, in the hypertrophied ventricular wall in chronic heart-disease fatty change is by no means infrequent. This degeneration may be limited to the heart or it may be more or less general in the solid viscera. The diaphragm may also be involved, even when the other muscles show no special changes. There appears to be a special proneness to fatty degeneration in the heart-muscle, which may perhaps be connected with its incessant activity. So great is its need of an abundant oxygen supply that it feels at once any deficiency, and is in consequence the first muscle to show nutritional changes.

Anatomically the condition may be local or general. The left ventricle is most frequently affected. If the process is advanced and general the heart looks large and is flabby and relaxed. It has a light yellowish-brown tint, or, as it is called, a faded-leaf color. Its consistence is reduced and the substance tears easily. In the left ventricle the papillary columns and the muscle beneath the endocardium show a streaked or

patchy appearance. Microscopically, the fibres are seen to be occupied by minute globules distributed in rows along the line of the primitive fibres (Welch). In advanced grades the fibres seem completely occupied by the minute globules.

(*b*) *Fatty Overgrowth.*—This is usually a simple excess of the normal subpericardial fat, to which the term *cor adiposum* was given by the older writers. In other instances the fat infiltrates the muscular substance and, separating the strands, may reach even to the endocardium. In corpulent persons there is always much pericardial fat. It forms part of the general obesity, and occasionally leads to dangerous or even fatal impairment of the contractile power of the heart. Of 122 cases analyzed by Forchheimer there were 88 males and 34 females. Over eighty per cent occurred between the fortieth and seventieth years.

The entire heart may be enveloped in a thick sheeting of fat through which not a trace of muscle substance can be seen. On section, the fat infiltrates the muscle, separating the fibres, and in extreme cases—particularly in the right ventricle—reaches the endocardium. In some places there may be even complete substitution of fat for the muscle substance. In rare instances the fat may be in the papillary muscles. The heart is usually much relaxed and the chambers are dilated. Microscopically the muscle fibres may show, in addition to the atrophy, marked fatty degeneration.

5. **Other Degenerations of the Myocardium.** (*a*) *Brown Atrophy.*—This is a common change in the heart-muscle, particularly in chronic valvular lesions and in the senile heart. When advanced, the color of the muscles is a dark red-brown, and the consistence is usually increased. The fibres present an accumulation of yellow-brown pigment chiefly about the nuclei. The cement substance is often unusually distinct, but seems more fragile than in healthy muscle.

(*b*) Amyloid degeneration of the heart is occasionally seen. It occurs in the intermuscular connective tissue and in the blood-vessels, not in the fibres.

(*c*) The hyaline transformation of Zenker is sometimes met with in prolonged fevers. The affected fibres are swollen, homogeneous, translucent, and the striæ are very faint or entirely absent.

(*d*) Calcareous degeneration may occur in the myocardium, and the muscle fibres may be infiltrated and yet retain their appearance as figured and described by Coats in his Text-book of Pathology.

Symptoms of Myocardial Disease.—These are notoriously uncertain. A man with advanced fibroid myocarditis may drop dead suddenly, while doing heavy work, without having complained of cardiac distress. On the other hand, a patient may present enfeebled, irregular action and signs of dilatation; he may have shortness of breath, œdema, and the general symptoms believed to be characteristic of cases of fibroid and fatty heart, and the post-mortem show little or no change in the myocardium.

Cardio-sclerosis or fibroid heart is in some cases characterized by a

feeble, irregular, slow pulse, with dyspnœa on exertion and occasional attacks of angina. Irregularity is present in many, but not in all cases. The pulse may be very slow, even 30 or 40 per minute. Ultimately the cases come under observation with the symptoms of cardiac insufficiency. The arrhythmia, which may have been present, becomes aggravated and, according to Riegel, may not only precede, but also persist after the cardiac insufficiency has passed away. This certainly does not hold in all cases, for a patient recently under observation had the most marked arrhythmia, which persisted after recovery from a severe attack of cardiac insufficiency in which he nearly died. Upon his return, a few weeks ago, with dilatation and arrhythmia, we agreed that the condition was probably one of cardio-sclerosis; but the autopsy showed simple dilatation without either fibroid or marked fatty change in the heart.

Fatty degeneration of the heart presents the same difficulties. Extreme fatty changes, as in pernicious anæmia, may be consistent with full, regular pulse and a regularly acting heart. In some of these cases the fat does not appear to interfere seriously with the function of the organ. The truth is it may exist in an extreme grade without producing symptoms, so long as great dilatation of the chambers does not occur. The cardiac irregularity, the dyspnœa, palpitation, and small pulse are in reality not symptoms of the fatty degeneration, but of dilatation which has supervened. The fatty *arcus senilis* is of no moment in the diagnosis of fatty heart. The heart-sounds may be weak and the action irregular. When dilatation occurs, there is often the gallop rhythm, shortening of the long pause, and a systolic murmur at the apex. Shortness of breath on exertion is an early feature in many cases, and anginal attacks may occur. There is sometimes a tendency to syncope, and in both fibroid and fatty heart there are attacks in which the patient feels cold and depressed and the pulse sinks to 40 or 30, or even, as in one case which I saw, to 26. The patient may wake from sleep in the early morning with an attack of severe cardiac asthma. These "spells" may be associated with nausea and may alternate with others in which there are anginal symptoms. These are the cases, too, in which for weeks there may be mental symptoms. The patient has delusions and may even become maniacal. Toward the close, Cheyne-Stokes breathing is met with in a number of cases.

Fatty overgrowth of the heart is a condition certain to exist in very obese persons. It produces no symptoms until the muscular fibre is so weakened that dilatation occurs. These patients may for years present a feeble but regular pulse; the heart-sounds are weak and muffled, and a murmur may be heard at the apex. Attacks of cardiac asthma are not uncommon, and the patient may suffer from bronchitis. Dizziness and pseudo-apoplectic seizures may occur. Sudden death may result from syncope or from rupture of the heart. The physical examination is often difficult because of the great increase in the fat, and it may be impossible to define the area of dulness.

For practical purposes we may group the cases of myocardial disease as follows:

(1) Those in which sudden death occurs with or without previous indications of heart-trouble. Sclerosis of the coronary arteries exists—in some instances with recent thrombus and white infarcts; in others, extensive fibroid disease; in others again, fatty degeneration. In many cases there is never any complaint of cardiac distress, but, as in the case of Chalmers, the celebrated Scottish divine, may enjoy unusual vigor of mind and body.

(2) Cases in which there are cardiac arrhythmia, shortness of breath on exertion, attacks of cardiac asthma, sometimes anginal attacks, collapse symptoms with sweats and extremely slow pulse, and occasionally marked mental symptoms. These are the cases in which the condition may be strongly suspected and, in some instances, diagnosed. It is rarely possible to make a distinction between the fatty and fibroid heart.

(3) Cases in which there are cardiac insufficiency and symptoms of dilatation of the heart. Dropsy is often present, and with a loud murmur at the apex it may be difficult, unless the case has been seen from the outset, to determine whether or not a valvular lesion is present.

Prognosis.—The outlook in affections of the myocardium is extremely grave. Patients recover, however, in a surprising way from the most serious attacks, particularly those of the second group.

Treatment.—Many cases never come under treatment; the first are the final symptoms.

Cases with signs of well-marked cardiac insufficiency, as manifested by dyspnœa, weak, irregular, rapid heart, and œdema, may be treated on the plan laid down for the treatment of broken compensation in valvular disease. Digitalis may be given even if fatty degeneration is suspected, and is often very beneficial.

Much more difficult is the management of those cases in which there is marked cardiac arrhythmia, with a feeble, irregular, very slow pulse, and syncope or angina. Dropsy is not, as a rule, present; the heart-sounds may be perfectly clear, and there are no signs of dilatation. Digitalis, under these circumstances, is not advisable, particularly when the pulse is infrequent. Complete rest in bed, a carefully regulated diet, and the use of the aromatic spirits of ammonia, sulphuric ether, and stimulants are indicated. For the restlessness and distressing feelings of anxiety morphia is invaluable. From an eightieth to a sixtieth of a grain of strychnia may be given three times a day. If, as is sometimes the case, the pulse is hard and firm, nitroglycerin may be cautiously administered, beginning with one minim of the one per cent solution three times a day and increased gradually.

In certain cases of weak heart, particularly when it is due to fatty overgrowth, the plan of treatment recommended by Oertl is advantageous. It is an invaluable method in those forms of heart-weakness due to intem-

perance in eating and drinking and defective bodily exercise. The Oertl plan consists of three parts. First, the reduction in the amount of liquid. This is an important factor in reducing the fat in these patients. It also slightly increases the density of the blood. Oertl allows daily about thirty-six ounces of liquid, which includes the amount taken with the solid food. Free perspiration is promoted by bathing (if advisable, the Turkish bath), or even by the use of pilocarpine.

The second important point in his treatment is the diet, which should consist largely of proteids.

Morning.—Cup of coffee or tea, with a little milk, about six ounces altogether. Bread, three ounces.

Noon.—Three to four ounces of soup, seven to eight ounces of roast beef, veal, game, or poultry, salad or a light vegetable, a little fish; one ounce of bread or farinaceous pudding; three to six ounces of fruit for dessert. No liquids at this meal, as a rule, but in hot weather six ounces of light wine may be taken.

Afternoon.—Six ounces of coffee or tea, with as much water. As an indulgence an ounce of bread.

Evening.—One or two soft-boiled eggs, an ounce of bread, perhaps a small slice of cheese, salad, and fruit; six to eight ounces of wine with four or five ounces of water (Yeo).

The most important element of all is graduated exercise, not on the level, but up hills of various grades. The distance walked each day is marked off and is gradually lengthened. In this way the heart is systematica· ·rcised and strengthened.

Ther· · doubt of the great value of this or like methods in appropriate cases. At several of the health resorts in Germany, particularly the Bad Nauheim, under Schott, the results are striking. The plan is rarely advisable in valvular lesions and should not be adopted when there is marked arterio-sclerosis. Cases of fatty overgrowth of the heart are those most suitable. The plan of treatment reduces the obesity, and the patients are, for a time at least, much more comfortable and are able to go about and do their work without cardiac distress or great shortness of breath.

Aneurism of the Heart.

(*a*) **Aneurism of a Valve** results from acute endocarditis, which produces softening or erosion and may lead either to perforation of the segment or to gradual dilatation of a limited area under the influence of the blood-pressure. The aneurisms are usually spheroidal and project from the ventricular face of a sigmoid valve. They are much less common on the mitral segments. They frequently rupture and produce extensive destruction and incompetency of the valves.

(*b*) **Aneurism of the Walls.**—This comparatively rare condition results from the weakening of the walls by chronic myocarditis, or occasionally

It follows mural endocarditis, which more commonly, however, leads to perforation. Aneurism has followed a stab-wound of the heart. The left ventricle near the apex is usually the seat, at the situation in which the fibrous degeneration is most common. Fifty-nine of the 90 cases collected by Legg were situated here. In the early stages the anterior wall of the ventricle, near the septum, sometimes involving the septum itself, is slightly dilated, the endocardium opaque, and the muscular tissue sclerotic. In a more advanced stage the dilatation is pronounced and layers of thrombi occupy the sac. Ultimately a large rounded tumor may project from the ventricle and may attain a size equal to that of the heart. Occasionally the aneurism is sacculated and communicates with the ventricle through a very small orifice. The sac may be double, as in a case reported by Janeway. In the museum of Guy's Hospital there is a specimen showing the wall of the ventricle covered with aneurismal bulgings. Rupture occurred in 7 of the 90 cases collected by Legg.

The *symptoms* produced by aneurism of the heart are indefinite. Occasionally there is marked bulging in the apex region and the tumor may perforate the chest wall. When the sac is large and produces pressure upon the heart itself, there may be a marked disproportion between the strong cardiac impulse and the feeble pulsation in the peripheral arteries.

Rupture of the Heart.

This rare event is usually associated with fatty infiltration or degeneration of the heart-muscles. In some instances, acute softening in consequence of embolism of a branch of the coronary artery, suppurative myocarditis, or a gummatous growth has been the cause. Of 100 cases collected by Quain, fatty degeneration was noted in 77. Two thirds of the patients were over sixty years of age.

The rent may occur in any of the chambers, but is found most frequently in the left ventricle on the anterior wall, not far from the septum. The accident usually takes place during exertion. There may be no preliminary symptoms, but without any warning the patient may fall and die in a few moments. Sudden death occurred in seventy-one per cent of Quain's cases. In other instances there may be in the cardiac region a sense of anguish and suffocation, and life may be prolonged for several hours. In a Montreal case which I examined the patient walked up a steep hill after the onset of the symptoms, and lived for thirteen hours. A case is on record in which the patient lived for eleven days.

New Growths and Parasites.

Tubercle and syphilis have already been considered. Primary cancer or sarcoma is extremely rare. Secondary tumors may be single or multiple, and are usually unattended with symptoms, even when the disease

is most extensive. In one case I found in the wall of the right ventricle a mass which involved the anterior segment of the tricuspid valve and partly blocked the orifice. The surface was eroded and there were numerous cancerous emboli in the pulmonary artery. In another instance the heart was greatly enlarged, owing to the presence of innumerable masses of colloid cancer the size of cherries. The mediastinal sarcoma may penetrate the heart, though it is remarkable how extensive the disease of the mediastinal glands may be without involvement of the heart or vessels.

Cysts in the heart are rare. They are found in different parts, and are filled either with a brownish or a clear fluid. Blood-cysts occasionally occur.

The parasites will be discussed under the appropriate section, but it may be mentioned here that both the *cysticercus cellulosæ* and the echinococcus cysts occur occasionally in the heart.

Wounds and Foreign Bodies.

Wounds of the heart are usually fatal, although there are many instances in which recovery has taken place. Bullets have been found encysted inside the ventricle. A majority of the cases of gunshot wounds, however, are necessarily fatal. Puncture of the heart by a sharp-pointed body, such as a needle or a stiletto, does not always prove fatal. Peabody has reported a case in which a pin was found embedded in the left ventricle. Suicide has been attempted by passing a needle or pin into the heart. It is not, however, necessarily fatal. Moxon mentioned a case, at the Clinical Society of London, in which a medical student, while on a spree, passed a pin into his heart. The pericardium was opened, and the head of the pin was found outside of the right ventricle. It was grasped and an attempt made to remove it, but it was withdrawn into the heart and, it is said, caused the patient no further trouble. Hysterical girls sometimes swallow pins and needles, which, passing through the œsophagus and stomach, are found in various parts of the body. A remarkable case is reported by Allen J. Smith of a girl from whom several dozen needles and pins were removed, usually from subcutaneous abscesses. Several years later she developed symptoms of chronic heart-disease. At the post-mortem needles were found in the tissues of the adherent pericardium, and between thirty and forty were embedded in the thickened pleural membranes of the left side.

Puncture of the heart has been recommended as a therapeutic procedure to stimulate it to action, as in chloroform narcosis, and experimental evidence has been brought forward by B. A. Watson in favor of the operation. He advises abstraction of blood in combination with the puncture—cardiocentesis. The proceeding is not without risk. Hæmorrhage may take place from the puncture, though it is not often extensive.

At the Philadelphia Hospital the procedure was tried by one of the resident physicians in a case of acute dilatation. The anterior coronary vein was cut across and considerable blood was found in the pericardium. There is danger also of striking Kronecker's inhibition centre.

V. NEUROSES OF THE HEART.

Palpitation.

In health we are unconscious of the action of the heart. In some people one of the first indications of debility or overwork is the consciousness of the cardiac pulsations, which may, however, be perfectly regular and orderly. This is not palpitation. The term is properly limited to irregular or forcible action of the heart perceptible to the individual.

Etiology.—The expression "perceptible to the individual" covers the essential element in palpitation of the heart. The most extreme disturbance of rhythm, a condition even of what is termed *delirium cordis*, may be unattended with subjective sensations of distress, and there may be no consciousness of disturbed action. On the other hand, there are cases in which complaint is made of the most distressing palpitation and sensations of throbbing, in which the physical examination reveals a regularly acting heart, the sensations being entirely subjective. We meet with this symptom in a large group of cases in which there is increased excitability of the nervous system. Palpitation may be a marked feature at the time of puberty, at the climacteric, and occasionally during menstruation. It is a very common symptom in hysteria and neurasthenia, particularly in the form of the latter which is associated with dyspepsia. Emotions, such as fright, are common causes of palpitation. It may occur as a sequence of the acute fevers. Females are more liable to the affection than males.

In a second group the palpitation results from the action upon the heart of certain substances, such as tobacco, coffee, tea, and alcohol. And, lastly, palpitation may be associated with organic disease of the heart, either of the myocardium or of the valves. As a rule, however, it is a purely nervous phenomenon—seldom associated with organic disease—in which the most violent action and the most extreme irregularity may exist without that subjective element of consciousness of the disturbance which constitutes the essential feature of palpitation.

The irritable heart described by Da Costa, which was so common among the young soldiers during the civil war, is a neurosis of this kind. The chief symptoms were palpitation with great frequency of the pulse on exertion, a variable amount of cardiac pain, and dyspnœa. The factors at work in producing this condition appeared to be the mental excitement, the unwonted muscular exertion associated with the drill, and diarrhœa.

The condition is not infrequent in civil life among young men, and it leads in some cases to hypertrophy of the heart.

Symptoms.—In the mildest form, such as occurs during a dyspeptic attack, there is slight fluttering of the heart and a sense of what patients sometimes call "goneness." In more severe attacks the heart beats violently, its pulsations against the chest wall are visible, the rapidity of the action is much increased, the arteries throb forcibly, and there is a sense of great distress. In some instances the heart's action is not at all quickened. The most striking cases are in neurasthenic women, in whom the mere entrance of a person into the room will cause the most violent action of the heart and throbbing of the peripheral arteries. The pulse may be rapidly increased until it reaches 150 or 160. A diffuse flushing of the skin may appear at the same time. After such attacks, there may be the passage of a large quantity of pale urine. In many cases of palpitation, particularly in young men, the condition is at once relieved by exertion. A patient with extreme irregularity of the heart may, after walking quickly one hundred yards or running up-stairs, return with the pulse perfectly regular. This is not infrequently seen, too, in the irregular action of the heart in mitral-valve disease.

The physical examination of the heart is usually negative. The sounds, the shock of which may be very palpable, are on auscultation clear, ringing, and metallic, but not associated with murmurs. The second sound at the base may be greatly accentuated. A murmur may sometimes be heard over the pulmonary artery or even at the apex in cases of rapid action in neurasthenia or in severe anæmia. The attacks may be transient, lasting only for a few minutes, or may persist for an hour or more. In some instances any attempt at exertion renews the attack.

The *prognosis* is usually good, though it may be extremely difficult to remove the conditions underlying the palpitation.

Arrhythmia.

An intermission occurs when one or more beats of the heart are dropped. Irregularity is the condition when the beats are unequal in volume and force, or follow each other at unequal distances. Allorrhythmia is a term which is also used to express deviations from the normal heart rhythm.

The following varieties of arrhythmical action may be recognized:

(1) The paradoxical pulse of Küssmaul, in which the beats during inspiration are more frequent but less full than during expiration. This is found in weak heart, in chronic pericarditis, and when fibrous bands encircle the root of the aorta; but it may also occur normally from the influence of the respirations upon the heart. It is sometimes to be felt in sleeping children.

(2) Intermittence, in which there is simply an intermission or drop-

ping of a cardiac beat. The term deficience is more correctly applied to those instances in which the absence of the heart-sound proves that the systole is really omitted. The systole may be so weak as not to produce a pulsation, and yet at the same time a feeble first sound may be heard.

(3) The alternate heart-beat, in which strong and weak pulsations alternate regularly and which is expressed in the peripheral arteries by alternate full and feeble pulse-beats.

(4) The bigeminal and trigeminal pulsations occur when two or three beats follow each other in rapid succession, each group being separated from the following by a longer interval. This is not very uncommon in mitral disease. In the bigeminal pulse the first beat of the pair is usually the stronger. Indeed, in the condition known as heart bigeminism the second systole is so feeble that the pulse wave does not reach the peripheral arteries and the two systoles are represented by only a single pulse-beat at the wrist.

(5) Delirium cordis, in which these various factors are combined and the heart's action is wholly irregular.

(6) Fœtal heart rhythm—embryocardia—described by Stokes, is a very common condition in which the long pause is shortened and the characters of the sounds are "almost completely identical." The resemblance to the fœtal heart-beat is very striking. In the later stages of fevers and in extreme dilatation this form of heart rhythm is very frequently heard.

(7) Gallop rhythm, in which the sounds resemble the footfall of a horse at canter, usually results from the reduplication of the second sound in a rapidly acting heart. It is expressed by the words "rat-ta-tat." Sometimes it seems as if the first sound was split; more commonly it is the second. It is most frequently heard in interstitial nephritis and arterio-sclerosis, but it is said to be met with also in healthy persons.

The causes of these various disturbances of rhythm are thus classified by G. Baumgarten: *

(1) Those due to central—cerebral—causes, either organic disease, as in hæmorrhage, or concussion; more commonly psychical influences.

(2) Reflex influences, such as produce the cardiac irregularity in dyspepsia and diseases of the liver, lungs, and kidneys.

(3) Toxic influences. Tobacco, coffee, and tea are common causes of arrhythmia. Various drugs, such as digitalis, belladonna, and aconite, may also induce it.

(4) Changes in the heart itself. (*a*) In the cardiac ganglia. Fatty, pigmentary, and sclerotic changes have been described in cases of this sort and may have an important influence in producing disturbances in the rhythm; but as yet we do not know their exact significance. They

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may be present in cases which have not presented arrhythmia. (*b*) Mural changes are common in conditions of this kind. Simple dilatation, fatty degeneration, and sclerosis are most commonly present, the two latter usually associated with sclerosis of the coronary arteries.

The significance of arrhythmia is not always easy to determine. Simple irregular action of the heart may persist for years. The late Chancellor Ferrier, of McGill University, a man of unusual bodily and mental vigor, who died at the age of eighty-seven, had an extremely irregular pulse for almost fifty years of his life. One or two other instances have come under my notice of persons in good health, without arterial or cardiac disease, in whom the heart's action was persistently irregular. The bigeminal and trigeminal pulsations are found more frequently in mitral than in other conditions. The delirium cordis is met with in the dilatation associated with valvular lesions, particularly toward the latter stages. Fœtal heart rhythm is rarely found apart from dilatation.

Rapid Heart—Tachycardia.

The rapid action may be perfectly natural. There are individuals whose normal heart action is at 100 or even more per minute. It may be caused by the various conditions which induce palpitation; but the two are not necessarily associated. Emotional causes, violent exercise, and fevers all produce great increase in the rapidity of the heart's action. The extremely rapid action which follows fright may persist for days, or even weeks. Traube reports an instance in which, after violent exercise, the rapid action of the heart continued. Cases are not uncommon at the menopause.

There are cases again in which the condition can hardly be termed a neurosis, since it depends upon definite changes in the pneumogastrics or in the medulla. Cases have been reported in which tumor or clot in or about the medulla or pressure upon the vagi has been associated with heart hurry. Some of the cases of frequent action of the heart in women have been thought to be due to reflex irritation from ovarian or uterine disease.

Paroxysmal tachycardia is a remarkable affection, characterized by spells of heart hurry, during which the action is greatly increased, the pulse reaching 200 and over. The cases are not common. The condition has been thoroughly studied by Nothnagel. The attack may be quite short and persist only for an hour or so. A patient at the Philadelphia Infirmary for Nervous Diseases was attacked every week or two; the pulse would rise to 220 or 230, and there were such feelings of distress and uneasiness that the patient always had to lie down. There may be, however, no subjective disturbance, and in another case the patient was able to walk about during the paroxysm and had no dyspnœa. One of the most remarkable cases is reported by H. C. Wood. A physician in his eighty-

seventh year has had attacks at intervals since his thirty-seventh year. The onset is abrupt and the pulse rapidly rises to 200 a minute. For more than twenty years the taking of ice-water or strong coffee would arrest the attacks. Bouveret has analyzed a number of cases of this essential or idiopathic form; he finds that a permanent cure is rare, and that the patients suffer for ten or more years. Four instances terminated fatally from heart-failure. Wood suggests that these cardiac paroxysms are caused by discharging lesions affecting the centres of the accelerator nerves. François Franck has shown that the acceleration of the heart's action is due to the shortening of the diastole, and during the systole so little blood is expelled from the heart that the average amount in the minute is not increased. Moreover, the accelerators appear to have no trophic relation to the heart, and stimulation of them is not accompanied either by increased arterial pressure or by augmentation of the work done by the heart.

Slow Heart—Brachycardia (*Bradycardia*).

Slow action of the heart is sometimes normal and may be a family peculiarity. Napoleon is stated to have had a pulse of only 40 per minute.

In any case of slow pulse it is important first to make sure that the number of heart and arterial beats correspond. In many instances this is not the case, and with a radial pulse at 40 the cardiac pulsations may be 80, half the beats not reaching the wrist. The heart contractions, not the pulse wave, should be taken into account. A most exhaustive study of this condition has been made recently by Riegel, whose division is here followed:

(*a*) Physiological brachycardia. In the puerperal state the pulse may beat from 44 to 60 per minute, or may even be as low as 34. It is seen in premature labor as well as at term. The explanation of its occurrence at this period is not clear. Slowness of the pulse is associated with hunger. Brachycardia depending on individual peculiarity is extremely rare.

(*b*) Pathological brachycardia, which is met with under the following conditions: (1) In convalescence from acute fevers. This is extremely common, particularly after pneumonia, typhoid fever, acute rheumatism, and diphtheria. It is most frequently seen in young persons and in cases which have run a normal course. Traube's explanation that it is due to exhaustion is probably the correct one. (2) In diseases of the digestive system, such as chronic dyspepsia, ulcer or cancer of the stomach, and jaundice. The largest number of Riegel's cases were of this group. (3) In diseases of the respiratory system. Here it is by no means so common, but is seen not infrequently in emphysema. (4) In diseases of the circulatory system. Excluding all cases of irregularity of the heart, brachycardia is not common in diseases of the valves. It is most frequently seen in fatty and fibroid changes in the heart, but is not constant in them. (5) In diseases of the urinary organs. It occurs occasionally in nephritis and

may be a feature of uræmia. (6) From the action of toxic agents. It occurs in uræmia, poisoning by lead, alcohol, and follows the use of tobacco, coffee, and digitalis. (7) In constitutional disorders, such as anæmia, chlorosis, and diabetes. (8) In diseases of the nervous system. Apoplexy, epilepsy, the cerebral tumors, affections of the medulla, and diseases and injuries of the cervical cord may be associated with very slow pulse. In general paresis, mania, and melancholia it is not infrequent. (9) It occurs occasionally in affections of the skin and sexual organs, and in sunstroke, or in prolonged exhaustion from any cause.

It is seen most frequently in the convalescence from acute fevers, then in disorders of the digestive system. The significance of this symptom is variable. It is only in diseases of the heart or brain that it is ominous. It may be due to direct irritation of the vagi, to diminished excitability of the cardiac ganglia, to reflex influences acting upon the vagus centre, or to weakness of the heart-muscle itself. The pulse-beat rarely sinks below 20. Prentice, at the Association of American Physicians at Washington, showed a patient with attacks of unconsciousness, who had, particularly during the attacks, but also in the intervals, a pulse as low as 12 per minute. Such cases are extremely rare. Cases are on record in which the pulse has fallen to 8 or 9 beats in the minute. At the discussion which followed the exhibition of Prentice's patient, both Jacobi and Kinnicutt referred to similar cases associated with epileptic seizures, in one of which the pulse fell as low as 7 in the minute.

Treatment of Palpitation and Arrhythmia.—An important element in many cases is to get the patient's mind quieted, and he can be assured that there is no actual danger. The mental element is oftentimes very strong. In palpitation, before using medicines, it is well to try the effect of hygienic measures. As a rule, moderate exercise may be taken with advantage. Regular hours should be kept, and at least ten hours out of the twenty-four should be spent in the recumbent posture. A tepid bath may be taken in the morning, or, if the patient is weakly and nervous, in the evening, followed by a thorough rubbing. Hot baths and the Turkish bath should be avoided. The dietetic management is most important. It is best to prohibit absolutely alcohol, tea, and coffee. The diet should be light and the patient should avoid taking large meals. Articles of food known to cause flatulency should not be used. If a smoker, the patient should give up tobacco. Sexual excitement is particularly pernicious, and the patient should be warned specially on this point. For the distressing attacks of palpitation which occur with neurasthenia, particularly in women, a rigid Weir-Mitchell course is the most satisfactory. It is in these cases that we find the most distressing throbbing in the abdomen, which is apt to come on after meals, and is very much aggravated by flatulency. The cases of palpitation due to excesses or to errors in diet and dyspepsia are readily remedied by hygienic measures.

A course of iron is often useful. Strychnia is particularly valuable, and is perhaps best administered as the tincture of nux vomica in large doses. Very little good is obtained from the smaller quantities. It should be given freely, 20 minims three times a day.

If there is great rapidity of action, aconite may be tried or veratrum viride. There are cases associated with sleeplessness and restlessness which are greatly benefited by bromide of potassium. Digitalis is very rarely indicated, but in obstinate cases it may be tried with the nux vomica.

Cases of heart hurry are often extremely obstinate, as may be judged from the case of the physician reported by H. C. Wood, in whom the condition persisted in spite of all measures for fifty years. The bromides are sometimes useful; the general condition of neurasthenia should be treated, and during the paroxysm an ice-bag may be placed upon the heart, or Leiter's coil, through which ice-water may be passed. Electricity, in the form of galvanism, is sometimes serviceable, and for its mental effect the Franklinic current. For the condition of slow pulse but little can be done. A great majority of the cases are not dangerous.

Angina Pectoris.

Stenocardia, or the breast-pang described by Heberden, is not an independent affection, but a symptom associated with a number of morbid conditions of the heart and vessels, more particularly with sclerosis of the root of the aorta and changes in the coronary arteries. True angina, which is a rare disease, is characterized by paroxysms of agonizing pain in the region of the heart, extending into the arms and neck. In violent attacks there is a sensation of impending death.

Etiology and Pathology.—It is a disease of adult life and occurs almost exclusively in men. Arterio-sclerosis, hypertrophy of the heart, increased arterial tension, or aortic insufficiency are often present, while anatomical changes in the aorta, arteries, and myocardium are almost constant. No instance of true angina has come under my observation in which there were not signs of cardio-vascular changes. The immediate exciting cause of an attack is most frequently sudden exertion or emotional excitement. The paroxysm may come on in the daytime, but in some of the worst cases they occur at night. The nature of the affection is doubtful. The following views have been entertained.

(1) That it is a neuralgia of the cardiac nerves. In the true form the agonizing cramp-like character of the pain, the suddenness of the onset, and the associated features, are unlike any neuralgic affection. The pain, however, is undoubtedly in the cardiac plexus and radiates to adjacent nerves. It is interesting to note in connection with the almost constant sclerosis of the coronary arteries in angina that Thoma has found marked sclerosis of the temporal artery in migraine and Dana has met with local thickening of the arteries in some cases of neuralgia (2) Heb-

erden believed that it was a cramp of the heart-muscle itself. This would explain the agonizing character of the pain and the suddenness of the onset as well as the frequency of the fatal termination; but if the cramps were general in the heart-muscle and similar to those which occur in the voluntary muscles, death would invariably ensue with great rapidity. Cramp of certain muscular territories would explain the attack. (3) That it is due to the extreme tension of the ventricular walls, in consequence of an acute dilatation associated, in the majority of cases, with affection of the coronary arteries. Traube, who supported this view, held that the agonizing pain resulted from the great stretching and tension of the nerves in the muscular substance. A modified form of this view is that there is a spasm of the coronary arteries with great increase of the intracardiac pressure.

In fatal cases of angina the coronary arteries are almost invariably diseased, either in their main division, or there is chronic endarteritis with great narrowing of the orifices at the root of the aorta. Experimentally, occlusion of the coronary arteries produces slowing of the heart's action, gradual dilatation, and death within a very few minutes. Cohnheim has shown that in the dog ligation of one of the large coronary branches produces within a minute a condition of arrhythmia, and within two minutes the heart ceases in diastole. These experiments, however, do not throw much light upon the etiology of angina pectoris. Extreme sclerosis of the coronary arteries is common, and a large majority of the cases present no symptoms of angina. Even in the cases of sudden death due to blocking of an artery, particularly the anterior branch of the coronary artery, there is usually no great pain either before or during the attack. The lesions of the nerves described by Lancereaux, Hadden, and others cannot yet be correlated satisfactorily with the symptoms of true angina. Various forms of true angina have been recognized, but the differences, in the majority of instances, are not sufficiently marked to permit a separation. Reference may be made, however, to the angina pectoris *vaso-motoria* described by Nothnagel. In this the attack may come on after exposure to cold. There is general spasm of the peripheral arteries with a sense of stiffness and deadness in the extremities, and pallor, cyanosis, and lowering of the temperature. The arteries are small and contracted. There is sometimes a feeling of faintness or even a loss of consciousness. With this there is a sense of pressure, tension, or even agonizing pain in the cardiac region. The pulse, however, is regular, and there are no signs of disease of the heart. The condition is supposed to depend upon a wide-spread spasm of the peripheral arteries. I have never recognized a case of this kind, although certain of its features are not at all uncommon in the pseudo-angina.

Symptoms.—Usually during exertion or intense mental emotion the patient is seized with an agonizing pain in the region of the heart and a sense of constriction, as if the heart had been seized in a vise. The pains

radiate up the neck and down the arm and there may be numbness of the fingers or in the cardiac region. The face is usually pallid and may assume an ashy-gray tint, and not infrequently a profuse sweat breaks out over the surface. Dyspnœa is not usually present. The paroxysm lasts from several seconds to a minute or two, during which, in severe attacks, the patient feels as if death were imminent. There is great restlessness and anxiety, and the patient may drop dead at the height of the attack or faint and pass away in syncope. The condition of the heart during the attack is variable; the pulsations may be uniform and regular. The pulse tension, however, is usually increased, but it is surprising, even in cases of extreme severity, how slightly the character of the pulse may be altered. After the attack there may be eructations, or the passage of a large quantity of clear urine. The patient usually feels exhausted, and for a day or two may be badly shaken; in other instances in an hour or two the patient feels himself again. The attacks may recur at intervals of a few weeks, or perhaps not for many years. There are individuals who have well-marked anginal attacks for years, and, except during the paroxysms, suffer but slight inconvenience.

Diagnosis.—There are many grades of true angina. A man may have slight præcordial pain, a sense of distress and uneasiness, and radiation of the pains to the arm and neck. Such attacks following slight exertion, an indiscretion in diet, or a disturbing emotion may alternate with attacks of much greater severity, or they may occur in connection with a pulse of increased tension and signs of general arterio-sclerosis. In the milder grades the diagnosis cannot rest upon the symptoms of the attack itself, since they may be simulated by the pseudo-angina; but the diagnosis should be based upon the examination of the circulatory system. In true angina, even in the milder forms, signs of arterio-sclerosis are usually present. In a case presenting attacks of præcordial pain or pains in the cervical or brachial plexuses, if the aortic second sound is clear, not ringing, the pulse tension low, and the peripheral arteries soft, the diagnosis of true angina should not be made. After all, the chief difficulty, however, arises in the cases of the hysterical or *pseudo-angina*.

This is a common affection in women, but may occur also in neurasthenic men. It is in this form particularly that we see vaso-motor phenomena. The patient may complain of great coldness of the hands or feet, or a general feeling of deadness and stiffness, often with pain in the back of the head and neck. The attacks recur frequently, and sometimes become worse at each monthly period. They may come on with great severity at the menopause. Worry and disturbing emotions of all kinds may at any time precipitate an attack. Huchard has given in concise form the following points in diagnosis between the true and hysterical angina:

TRUE ANGINA.	PSEUDO-ANGINA.
Most common between the ages of forty and fifty years.	At every age, even six years.
Most common in men. Attacks brought on by exertion.	Most common in women. Attacks spontaneous.
Attacks rarely periodical or nocturnal.	Often periodical and nocturnal.
Not associated with other symptoms.	Associated with nervous symptoms.
Vaso-motor form rare. Agonizing pain and sensation of compression by a vise.	Vaso-motor form common. Pain less severe; sensation of distention.
Pain of short duration. Attitude: silence, immobility.	Pain lasts one or two hours. Agitation and activity.
Lesions: sclerosis of coronary artery.	Neuralgia of nerves and cardio-plexus.
Prognosis grave, often fatal.	Never fatal.
Arterial medication.	Antineuralgic medication.

There are cases in women which are sometimes very puzzling; for instance, when the patient presents a combination of marked hysterical manifestations and attacks of angina and has aortic insufficiency. In such instances the patient should receive the benefit of the doubt and be treated for true angina.

Prognosis.—Cardiac pain without evidence of arterio-sclerosis or valve disease is not of much moment. True angina is almost invariably associated with marked cardio-vascular lesions in which the prognosis is always grave. With judicious treatment the attacks, however, may be long deferred, and a few instances recover completely. The prognosis is naturally more serious with aortic insufficiency and advanced arterio-sclerosis. Patients who have had well-marked attacks may live for many years, but much depends upon the care with which they regulate their daily life.

Treatment.—Patients subject to this affection should live a quiet life, avoiding particularly excitement and sudden muscular exertion. During the attack nitrite of amyl should be inhaled, as advised by Lauder Brunton. From two to five drops may be placed upon cotton-wool in a tumbler or upon the handkerchief. This is frequently of great service in the attack, relieving the agonizing pain and distress. Subjects of the disease should carry the *perles* of the nitrite of amyl with them, and use them on the first indication of an attack. In some instances the nitrite of amyl is quite powerless, though given freely. If within a minute or two relief is not obtained in this way, chloroform should at once be given. A few inhalations act promptly and give great relief. Should the pains continue, a hypodermic of morphia may be administered.

In the intervals, nitroglycerin may be given in full doses, as recommended by Murrell, or the nitrite of sodium (Matthew Hay). The nitroglycerin should be used for a long time and in increasing doses, beginning with one minim three times a day of the one per cent solution, and increasing the dose one minim every five or six days until the patient complains of flushing or headache.

Huchard recommends the iodides, believing that their prolonged use influences the arterio-sclerosis. Twenty grains three times a day may be given for several years, omitting the medicine for about ten days in each month. In some instances this treatment is certainly beneficial. Two men, both with arterio-sclerosis, ringing, accentuated aortic sound, and attacks of true angina, have under its use remained practically free from attacks—one case for nearly three, and the other for fully four years. This treatment is, however, not always satisfactory, and I have had several cases in which the condition has not been at all relieved by it.

For the pseudo-angina, the treatment must be directed to the general nervous condition. Electricity is sometimes very beneficial, particularly the Franklinic form.

VI. CONGENITAL AFFECTIONS OF THE HEART.

These have only a limited clinical interest, as in a large proportion of the cases the anomaly is not compatible with life, and in others nothing can be done to remedy the defect or even to relieve the symptoms.

The congenital affections result from interruption of the normal course of development or from inflammatory processes—endocarditis; sometimes from a combination of both.

(*a*) Of *general anomalies* of development the following conditions may be mentioned: *Acardia*, absence of the heart, which has been met with in the monstrosity known by the same name; *double heart*, which has occasionally been found in extreme grades of fœtal deformity; *dextrocardia*, in which the heart is on the right side, either alone or as part of a general transposition of the viscera; *ectopia cordis*, a condition associated with fission of the chest wall and of the abdomen. The heart may be situated in the cervical, pectoral, or abdominal regions. Except in the abdominal variety the condition is very rarely compatible with extra-uterine life.

(*b*) **Anomalies of the Cardiac Septa.**—The septa of both auricles and ventricles may be defective, in which case the heart consists of but two chambers, the *cor biloculare* or reptilian heart. In the septum of the auricles there is a very common defect, owing to the fact that the membrane closing the foramen ovale has failed at one point to become attached to the ring, and leaves a valvular slit which may be large enough to admit the

handle of a scalpel. Neither this nor the small cribriform perforations of the membrane are of any significance.

The foramen ovale may be patent without a trace of membrane closing it. In some instances this exists with other serious defects, such as stenosis of the pulmonary artery, or imperfection of the ventricular septum. In others the patent foramen ovale is the only anomaly, and in many instances it does not appear to have caused any embarrassment, as the condition has been found in persons who have died of various affections. The ventricular septum may be absent, the condition known as trilocular heart. Much more frequently there is a small defect in the upper portion of the septum, either in the situation of the membranous portion known as the "undefended space" or in the region situated just anterior to this. The anomaly is very frequently associated with narrowing of the pulmonary orifice or of the conus arteriosus of the right ventricle.

(*c*) **Anomalies and Lesions of the Valves.**—Numerical anomalies of the valve are not uncommon. The semilunar segments at the arterial orifices are not infrequently increased or diminished in number. Supernumerary segments are more frequent in the pulmonary artery than in the aorta. Four, or sometimes five, valves have been found. The segments may be of equal size, but, as a rule, the supernumerary valve is small.

Instead of three there may be only two semilunar valves, or, as it is termed, the *bicuspid condition*. In my experience, this is most frequent in the aortic valve. Of twenty-one instances only two occurred at the pulmonary orifice. Two of the valves have united, and from the ventricular face show either no trace of division or else a slight depression indicating where the union had occurred. From the aortic side there is usually to be seen some trace of division into two sinuses of Valsalva. There has been a discussion as to the origin of this condition, whether it is really an anomaly or whether it is not due to endocarditis, fœtal or post-natal. The combined segment is usually thickened, but the fact that this anomaly is met with in the fœtus without a trace of sclerosis or endocarditis shows that it may, in some cases at least, result from a developmental error.

Clinically this is a very important congenital defect, owing to the liability of the combined valve to sclerotic changes. Except two fœtal specimens all of my cases showed thickening and deformity, and in fifteen of those which I have reported death resulted directly or indirectly from the lesion.

The little fenestrations at the margins of the sigmoid valves have no significance; they occur in a considerable proportion of all bodies.

Anomalies of the auriculo-ventricular valves are not often met with.

Fœtal endocarditis may occur either at the arterial or auriculo-ventricular orifices. It is nearly always of the chronic or sclerotic variety. Very rarely indeed is it of the warty or verrucose form. There are little nodular bodies, sometimes six or eight in number, on the mitral and tricuspid segments—the nodules of Albini—which represent the remains of

fœtal structures, and must not be mistaken for endocardial outgrowths. The little rounded, bead-like hæmorrhages of a deep purple color, which are very common on the heart valves of children, are also not to be mistaken for the products of endocarditis. In fœtal endocarditis the segments are usually thickened at the edges, shrunken, and smooth. In the mitral and tricuspid valves the cusps are found united and the chordæ tendineæ are thickened and shortened. In the semilunar valves all trace of the segments has disappeared, leaving a stiff membranous diaphragm perforated by an oval or rounded orifice. It is sometimes very difficult to say whether this condition has resulted from fœtal endocarditis or whether it is an error in development. In very many instances the processes are combined; an anomalous valve becomes the seat of chronic sclerotic changes, and, according to Rauchfuss, endocarditis is more common on the right side of the heart only because the valves are here most often the seat of developmental errors.

Lesions at the Pulmonary Orifice.—*Stenosis* of this orifice is one of the commonest and most important of congenital heart affections. A slow endocarditis causes gradual union of the segments and narrowing of the orifice to such a degree that it only admits the smallest-sized probe. In some of the cases the smooth membranous condition of the combined segments is such that it would appear to be the result of faulty development. In some instances vegetations develop. The condition is compatible with life for many years, and in a considerable proportion of the cases of heart-disease above the tenth year this lesion is present. With it there may be defect of the ventricular septum. Obliteration or *atresia* of the pulmonary orifice is less frequent but a more serious condition than stenosis. It is of necessity associated with either imperfection of the ventricular septum or patency of the foramen ovale and persistence of the ductus arteriosus. *Stenosis of the conus arteriosus* of the right ventricle exists in a considerable proportion of the cases of obstruction at the pulmonary orifice. At the outset a developmental error, it may be combined with sclerotic changes. The ventricular septum is imperfect, the foramen ovale is usually open, and the ductus arteriosus patent. These three lesions at the pulmonary orifice constitute the most important group of all congenital cardiac affections. Of 181 instances of various congenital anomalies collected by Peacock 119 cases came under this category, and, according to this author, in eighty-six per cent of the patients with congenital heart-disease living beyond the twelfth year the lesion is at this orifice.

Congenital lesions of the aortic orifice are not very frequent. Rauchfuss has collected 24 cases of stenosis and atresia, and stenosis of the left conus arteriosus may also occur, a condition which is not incompatible with prolonged life. Ten of the sixteen cases tabulated by Dilg were over thirty years of age.

Symptoms of Congenital Heart-disease.—Cyanosis occurs in over ninety per cent of the cases and forms so distinctive a feature that

the terms "blue disease" and "morbus cæruleus" are practically synonyms for congenital heart-disease. The lividity in a majority of cases appears early, within the first week of life, and may be general or confined to the lips, nose, and ears, and to the fingers and toes. In some instances there is in addition a general dusky suffusion, and in the most extreme grades the skin is almost purple. It may vary a good deal and may only be intense on exertion. The external temperature is low. Dyspnœa on exertion and cough are common symptoms. The children rarely thrive and often display a lethargy of both mind and body. The fingers and toes are clubbed in a grade rarely met with in any other affection. The cause of the cyanosis has been much discussed. Morgagni referred it to the general congestion of the venous system due to obstruction, and this view was supported in a paper, one of the ablest that has been written on the subject, by Moreton Stillé. Morrison's recent analysis of 75 cases of congenital heart-disease shows that closure of the pulmonary orifice and patency of the foramen ovale and the ventricular septum are the lesions most frequently associated with cyanosis, and he concludes that the deficient aëration of the blood owing to diminished lung function is the most important factor. Another view, advocated by William Hunter, was that the discoloration was due to the admixture in the heart of venous and arterial blood; but lesions may exist which permit of very free mixture without producing cyanosis.

Diagnosis.—In the case of children, cyanosis, with or without enlargement of the heart, and the existence of a murmur are sufficient, as a rule, to determine the presence of a congenital heart-lesion. The cyanosis gives us no clew to the precise nature of the trouble, as it is a symptom common to many lesions and it may be absent in certain conditions. The murmur is usually systolic in character. It is, however, not always present, and there are instances on record of complicated congenital lesions in which the examination showed normal heart-sounds. In two or three instances fœtal endocarditis has been diagnosed *in gravida* by the presence of a rough systolic murmur, and the condition has been corroborated subsequent to the birth of the child. Hypertrophy is present in a majority of the cases of congenital defect. It is impossible in the scope of a work of this sort to enter upon elaborate details in differential diagnosis between the various congenital heart-lesions. I here abstract the conclusions on this question given by Hochsinger in his recent monograph:*

"(1) In childhood, loud, rough, musical heart-murmurs, with normal or only slight increase in the heart-dulness, occur only in congenital heart-disease. The acquired endocardial defects with loud heart-murmurs in young children are almost always associated with great increase in the heart-dulness.

"(2) In young children heart-murmurs with great increase in the

* Die Auscultation des kindlichen Herzens, Wien, 1890.

cardiac dulness and feeble apex beat suggest congenital changes. The increased dulness is chiefly of the right heart, whereas the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex beat is visible; the dilatation of the right heart comes late and does not materially change the increased strength of the apex beat.

"(3) The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

"(4) An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which in early childhood is only to be explained by the assumption of a congenital pulmonary stenosis, and possesses therefore an importance from a point of differential diagnosis which is not to be underestimated.

"(5) Absence of a palpable thrill, despite loud murmurs which are heard over the whole præcordial region, is rare except with congenital defects in the septum, and it speaks therefore against an acquired cardiac affection.

"(6) Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important for the diagnosis of a persistence of the ductus Botalli, and cannot be explained by the assumption of an endocarditis of the aortic valve."

Treatment.—The child should be warmly clad and guarded from all circumstances liable to excite bronchitis. In the attacks of urgent dyspnœa with lividity blood should be freely let. Saline cathartics are also useful. Digitalis must be used with care, and it is sometimes beneficial in the later stages. When the compensation fails, the indications for treatment are those of valvular disease in adults.

III. DISEASES OF THE ARTERIES.

I. DEGENERATIONS.

Fatty degeneration of the intima is extremely common, and is seen in the form of yellowish-white spots in the aorta and larger vessels. *Calcification* of the arterial wall follows fatty degeneration, atheromatous changes, and sclerosis. It occurs in either the intima or the media. In the latter it produces what is sometimes known as annular calcification, which occurs particularly in the middle coat of medium-sized vessels and may convert them into firm tubes. Calcification of the intima is a common terminal process of arterio-sclerosis.

Hyaline degeneration may attack either the larger or the smaller vessels. In the former the intima is converted into a smooth, homogeneous substance, and it is commonly an initial stage of arterio-sclerosis. In the smaller arteries and capillaries the hyaline degeneration is often seen, particularly in the glomeruli of the kidney. Its exact production is still a matter of some doubt. "It appears to arise principally by homogeneous coagulation of an albuminous fluid, either within the vessels or infiltrating the cells and the hyaline transformation of proliferating cells and of leucocytes."

II. ARTERIO-SCLEROSIS (*Arterio-capillary Fibrosis*).

The conception of arterio-sclerosis as an independent affection—a general disease of the vascular system—is due to Gull and Sutton.

Definition.—A condition of thickening, diffuse or circumscribed, of the intima, consequent upon primary changes in the media and adventitia. The process leads, in the larger arteries, to what is known as atheroma or endarteritis deformans.

Etiology.—(1) As an involution process arterio-sclerosis is an accompaniment of old age, and is the expression of the natural wear and tear to which the tubes are subjected. Longevity is a vascular question, and has been well expressed in the axiom that "a man is only as old as his arteries." To a majority of men death comes primarily or secondarily through this portal. The onset of what may be called physiological arterio-sclerosis depends, in the first place, upon the quality of arterial tissue (vital rubber) which the individual has inherited, and secondly upon the amount of wear and tear to which he has subjected it. That the former plays the most important *rôle* is shown in the cases in which arterio-sclerosis sets in early in life in individuals in whom none of the recognized etiological factors can be found. Thus, for instance, a man of twenty-eight or twenty-nine may have arteries of sixty, and a man of forty may present vessels as much degenerated as they should be at eighty. Entire families sometimes show this tendency to early arterio-sclerosis, a tendency which cannot be explained in any other way than that in the make-up of the machine bad material was used for the tubing.

More commonly the arterio-sclerosis results from the bad use of good vessels, and among the circumstances which tend to produce this condition are the following:

(2) *Chronic Intoxications.*—Alcohol, lead, gout, and syphilis play an important *rôle* in the causation of arterio-sclerosis, although the precise mode of their action is not yet very clear. They may act, as Traube suggests, by increasing the peripheral resistance in the smaller vessels and in this way raising the blood tension, or possibly, as Bright taught, they alter

the quality of the blood and render more difficult its passage through the capillaries.

The poison of syphilis and of gout may act directly on the arteries, producing degenerative changes in the media and adventitia.

(3) *Overeating.*—Many authors attribute an important part of the etiology of arterio-sclerosis to the overfilling of the blood-vessels which occurs when unnecessarily large quantities of food and drink are taken. Particularly is this the case in stout persons who take very little exercise.

(4) *Overwork of the muscles*, which acts by increasing the peripheral resistance and by raising the blood-pressure.

(5) *Renal Disease.*—The relation between the arterial and kidney lesions has been much discussed, some regarding the arterial degeneration as secondary, others as primary. There are certainly two groups of cases, one in which the arterio-sclerosis is the first change, and the other in which it appears to be secondary to a primary affection of the kidneys. The former occurs, I believe, with much greater frequency than has been supposed.

Morbid Anatomy.—Thoma divides the cases into *primary* arterio-sclerosis, in which there are local changes in the arteries leading to dilatation and a compensatory increase of the connective tissue of the intima; *secondary* arterio-sclerosis, due to changes in the arteries which follow increased resistance to the blood-flow in the peripheral vessels. This increased tension leads to dilatation and to slowing of the blood-stream and a secondary compensatory development of the intima.

In a recent study of 41 autopsies upon arterio-sclerotic cases from my wards, Councilman * follows the useful division into nodular, senile, and diffuse forms.

(*a*) *Nodular Form.*—In the circumscribed or nodular variety the macroscopic changes are very characteristic. The aorta presents, in the early stages, from the ring to bifurcation, numerous flat projections, yellowish or yellowish white in color, hemispherical in outline, and situated particularly about the orifices of the branches. In the early stage these patches are scattered and do not involve the entire intima. In more advanced grades the patches undergo atheromatous changes. The material constituting the button undergoes softening and breaks up into granular material, consisting of molecular *débris*—the so-called atheromatous abscess.

In the circumscribed or nodular arterio-sclerosis the primary alteration consists in a degeneration or a local infiltration in the media and adventitia, chiefly about the vasa vasorum. The affection is really a mesarteritis and a periarteritis. These changes lead to the weakening of the wall in the affected area, at which spot the proliferative changes commence in the intima, particularly in the subendothelial structures, with gradual thick-

* Transactions of the Association of American Physicians, vol. vi.

ening and the formation of an atheromatous button or a patch of nodular arterio-sclerosis. The researches of Thoma have shown that this is really a compensatory process, and that before its degeneration the nodular button, which post mortem projects beyond the lumen, during life fills up and obliterates what would otherwise be a depression of the wall in consequence of the weakening of the media. A similar process goes on in the smaller vessels, and in any one of the smaller branches it can be readily seen on section that each patch of endarteritis corresponds to a defect in the media and often to changes in the adventitia. The condition is one which may lead to rapid dilatation or to the production of an aneurism, particularly in the early stage, before the weakened spot is thickened and strengthened by the intimal changes.

(*b*) *Senile Arterio-sclerosis.*—The larger arteries are dilated and tortuous, the walls thin but stiff, and often converted into rigid tubes. The subendothelial tissue undergoes degeneration and in spots breaks down, forming the so-called atheromatous abscesses, the contents of which consist of a molecular *débris*. They may open into the lumen, when they are known as atheromatous ulcers. The greater portion of the intima may be occupied by rough calcareous plates, with here and there fissures and losses of substance, upon which not infrequently white thrombi are deposited. Microscopically there is extreme degeneration of the coats, particularly of the media. Senile atrophy of the liver and kidneys usually accompanies these changes. Senile changes are common in other organs. The heart may be small and is not necessarily hypertrophied. In 7 of 14 cases of Councilman's series there was no enlargement. Brown atrophy is common

(*c*) *Diffuse Arterio-sclerosis.*—The process is wide-spread throughout the aorta and its branches, in the former usually, but not necessarily, associated with the nodular form. The subjects of this variety are usually middle-aged men, but it may occur early. Of the 27 in Councilman's series belonging to this group the majority were between the ages of forty and fifty-five. The youngest was a negro of twenty-three and the oldest a man of sixty. The affection is very prevalent among negroes; less than fifty per cent were in whites, whereas the ratio of colored to white patients in the wards is one to seven. The affection is met with in strongly built, muscular men and, as Councilman remarks, they rarely present on the autopsy table signs of general anasarca or, if œdema exists, it has come on during the last few days of life.

The aorta and its branches are more or less dilated, the branches sometimes more than the trunk. The intima may be smooth and show very slight changes to the naked eye; more commonly there are scattered elevated areas of an opaque white color, some of which may have undergone atheromatous changes as in the senile form. Microscopically the *media* shows necrotic and hyaline changes, involving in the larger arteries both muscular and elastic elements, and the *intima* presents a great increase

in the subendothelial connective tissue, which is particularly marked opposite areas of advanced degeneration in the media. The small arteries—those of the kidneys, for example—show "a thickening of the wall, due to the formation of a homogeneous hyaline tissue within the muscular coat. This tissue contains but few cells, is faintly striated, and stains a light brown in the osmic acid used in the hardening solution. In many of the smallest vessels nothing can be seen of the elastic lamina, in others only fragments can be made out, in others it is preserved. . . . The muscular fibres of the media show marked atrophic changes. Fatty degeneration of the cells can be made out both in fresh sections and after hardening in Fleming's solution. The nuclei are thin and atrophic and vacuoles are sometimes seen in them. In some arteries the muscle-fibres have almost disappeared and the media is changed into a homogeneous tissue, similar to that in the thickened intima" (Councilman). The degeneration of the media is most marked in the smaller arteries. The capillaries are thickened, particularly those of the glomeruli of the kidneys, which are often obliterated and involved in extensive hyaline degeneration.

It is in this group of cases that the heart shows the most important changes. The average weight in the cases referred to was over 450 grammes, and there were two cases in which without valvular disease the weight was over 800 grammes. Fibrous myocarditis is often present, particularly when the coronary arteries are involved. The semilunar valves are sometimes opaque and sclerotic, and may be incompetent. The kidneys may show extensive sclerosis, but in many cases the changes are so slight that macroscopically they might be overlooked. They may be increased in size. The capsule is usually adherent, the surface a little rough, and very often presents atrophic areas at a lower level of a deeper red color. Increased consistence is always present.

Sclerosis of the pulmonary artery is met with in all conditions which for a long time increase the tension in the lesser circulation, particularly in mitral-valve disease and in emphysema. Sometimes the sclerosis reaches a high grade and is accompanied with aneurismal dilatation of the primary and secondary branches, more rarely with insufficiency of the pulmonary valve. In a remarkable case of a young man of twenty-four, reported by Romberg from Curschmann's clinic, the pulmonary arteries were involved in most extensive arterio-sclerosis; the main branches were dilated, and the smaller branches were the seat of the most extreme sclerotic changes. On the other hand, the aorta and its branches were normal. The heart was greatly hypertrophied, and the clinical symptoms were those of a congenital heart affection. In many cases of arterio-sclerosis the condition is not confined to the arteries, but extends not only to the capillaries but also to the veins, and may properly be termed *angio-sclerosis*.

Sclerosis of the veins—*phlebo-sclerosis*—is not at all an uncommon accompaniment of arterio-sclerosis, and is a condition to which of late a good deal of attention has been paid. It is seen in conditions of height-

ened blood-pressure, as in the portal system in cirrhosis of the liver and in the pulmonary veins in mitral stenosis. The affected vessels are usually dilated, and the intima shows, as in the arteries, a compensatory thickening, which is particularly marked in those regions in which the media is thinned. The new-formed tissue in the endophlebitis may undergo hyaline degeneration, and is sometimes extensively calcified. In a case of fibroid obliteration of the portal vein of long standing, I found the intima of the greatly dilated gastric, splenic, and mesenteric extensively calcified. In ordinary diffuse arterio-sclerosis the veins may also be involved, but rarely to a marked degree.

Symptoms.—Many patients never come under observation during life, but are seen for the first time on the post-mortem table, having died suddenly from cerebral hæmorrhage, blocking of a coronary artery, or rupture of an aneurism.

Among important symptoms are the following:

Increased Tension.—The pressure with which the blood flows in the arteries depends upon the degree of peripheral resistance and the force of the ventricular contraction. A high-tension pulse may exist with very little arterio-sclerosis; but, as a rule, when the condition has been persistent, the sclerosis and high tension are found together. The pulse wave is slow in its ascent, enduring, subsides slowly, and in the intervals of the beats the vessel remains full and firm. It may be very difficult to obliterate the pulse, and the firmest pressure on the radial or the temporal may not be sufficient to annihilate the pulse wave beyond the point of pressure. The sphygmographic tracing shows a sloping, short upstroke, no percussion wave, and a slow, gradual descent, in which the dicrotic wave is very slightly marked. It may be difficult to estimate how much of the hardness and firmness is due to the tension of the blood within the vessel, and how much to the thickening of the wall. If, for example, when the radial is compressed with the index-finger the artery can be felt beyond the point of compression, its walls are sclerosed.

Hypertrophy of the Heart.—In consequence of the peripheral resistance and increased work the left ventricle increases in size, and some of the purest examples of simple hypertrophy occur in this condition. The chamber may be little, if at all, dilated. The apex beat is dislocated in advanced cases an inch or more beyond the nipple line. The impulse is heaving and forcible. The aortic second sound is clear, ringing, and accentuated.

The combination of increased arterial tension, a palpable thickening of the arteries, hypertrophy of the left ventricle, and accentuation of the aortic second sound are signs pathognomonic of arterio-sclerosis. From this period of establishment the course of the disease may be very varied. For years the patient may maintain good health, and be in a condition analogous to a person with a well-compensated valvular lesion. There may be no renal symptoms, or there may be the passage of a larger

amount of urine than normal, with transient albuminuria, and now and then hyaline tube-casts. The subsequent history is extraordinarily diverse, depending upon the vascular territory in which the sclerosis is most advanced, or upon the accidents which are so liable to happen, and the symptoms may be cardiac, cerebral, renal, etc.

(1) *Cardiac.*—The involvement of the coronary arteries may lead to the various symptoms already referred to under that section—thrombosis with sudden death, fibroid degeneration of the heart, aneurism of the heart, rupture, and angina pectoris. Angina pectoris is extremely common, and in the true variety is almost always associated with arterio-sclerosis. A second important group of cardiac symptoms results from the dilatation which ultimately may follow the hypertrophy. The patient then presents all the symptoms of cardiac insufficiency—dyspnœa, scanty urine, and very often serous effusions. If the case has come under observation for the first time the clinical picture is that of chronic valvular disease, and the existence of a loud blowing murmur at the apex may throw the practitioner off his guard. Many cases terminate in this way.

(2) The *cerebral* symptoms of arterio-sclerosis are varied and important, and embrace those of many degenerative processes, acute and chronic (which follow sclerosis of the smaller branches), and cerebral hæmorrhage, which is usually associated with the miliary aneurisms.

Transient hemiplegia, monoplegia, or aphasia may occur in advanced arterio-sclerosis. Recovery may be perfect. It is difficult to say upon what these attacks depend. Spasm of the arteries has been suggested, but the condition of the smaller arteries is not very favorable to this view. Peabody has recently called attention to these cases, which are more common than indicated in the literature.

(3) *Renal* symptoms supervene in a large number of the cases. A sclerosis, patchy or diffuse, is present in a majority of the cases at the time of autopsy, and the condition is practically that of contracted kidneys. It is seen in a typical manner in the senile form, and not infrequently develops early in life as a direct sequence of the diffuse variety. It is often difficult to decide clinically (and the question is one upon which good observers might not agree in a given case) whether the arterial or the renal disease has been primary.

(4) Among other events in arterio-sclerosis may be mentioned gangrene of the extremities, due either directly to endarteritis or to the dislodgment of thrombi. Respiratory symptoms are not uncommon, particularly bronchitis and the symptoms associated with emphysema.

Treatment.—In the late stages the conditions must be treated as they arise in connection with the various viscera. In the early stages, before any local symptoms are manifest, the patient should be enjoined to live a quiet, well-regulated life, avoiding excesses in food and drink. It is usually best to explain frankly the condition of affairs, and so gain his intelligent co-operation. Special attention should be paid to the state of

the bowels and urine, and the secretion of the skin should be kept active by daily baths. Alcohol in all forms should be prohibited, and the food should be restricted to plain, wholesome articles. The use of mineral waters or a residence every year at one of the mineral springs is usually serviceable. If there has been a syphilitic history an occasional course of iodide of potassium is indicated, and whenever the pulse tension is high nitroglycerine may be used.

In cases which come under observation for the first time with dyspnœa, slight lividity, and signs of cardiac insufficiency, venesection is indicated. In some instances, with very high tension, striking relief is afforded by the abstraction of twenty ounces of blood.

III. ANEURISM.

The following forms of aneurism are usually recognized:

(*a*) The *true*, in which the sac is formed of one or more of the arterial coats. This may be fusiform, cylindrical, or cirsoid (in which the dilatation is in an artery and its branches), or it may be circumscribed or sacculated. Aneurisms are usually fusiform, resulting from uniform dilatation of the vessel, or saccular.

(*b*) The *false* or *dissecting* aneurism, which results from injury or laceration of the internal coat. The blood dissects between the layers; hence the name, dissecting aneurism. This occurs usually in the aorta. It may dissect the entire length of the vessel, and, perforating into the lumen of the vessel, may, as in a case reported by J. E. Graham, persist for years.

(*c*) *Arterio-venous* aneurism results when a communication is established between an artery and a vein. A sac may intervene, in which case it is called a varicose aneurism; but in many cases the communication is direct and the chief change is in the vein, which is dilated, tortuous, and pulsating, and is termed an aneurismal varix.

Etiology and Pathology.—Aneurisms arise: (*a*) By the gradual diffuse distention of the arterial coats, which have been weakened by arterio-sclerosis, particularly in its early stages, before compensatory endarteritis develops. The arch of the aorta is often dilated in this way so as to form an irregular aneurism.

(*b*) In consequence of circumscribed loss of resisting power in the media and adventitia, and due often to laceration of the media. This is the most common cause of sacculated aneurism. The laceration is frequently found in the ascending portion of the arch and occurs early in the process of arterio-sclerosis, before the compensatory thickening has taken place. Occasionally one meets with remarkable specimens illustrating the important part played by this process. The intima may also be torn. In a case of Daland's there was just above the aortic valves

an old transverse tear of the intima, extending almost the entire circumference of the vessel. Sclerosis of the media and adventitia had taken place and the process was evidently of some standing. An inch or more above it was a fresh transverse rent which had produced a dissecting aneurism. These arterio-sclerotic aneurisms, as they are called, are found also in the smaller vessels.

(*c*) *Embolic Aneurism.*—When an embolus has lodged in a vessel and permanently plugged it, aneurismal dilatation may follow on the proximal side. The embolus itself may, if a calcified fragment from a valve, lacerate the wall, or if infected may produce inflammation and softening. In either case aneurism may result.

(*d*) *Mycotic Aneurism.*—The importance of this form has been specially considered by Eppinger in his exhaustive monograph. The occurrence of multiple aneurisms in malignant endocarditis has been observed by several writers. Probably the first case in which the mycotic nature was recognized was one which occurred at the Montreal General Hospital and is reported in full in my lectures on malignant endocarditis. In addition to the ulceration of the valves there were four aneurisms of the arch, of which one was large and saccular, and three were not bigger than cherries. An extensive growth of micrococci was present in the larger as well as in the smaller sacs.

A form of parasitic aneurism which occurs with great frequency in the mesenteric arteries of the horse is due to the development of the *strongylus armatus*.

And, lastly, there are cases in which without any definite cause there is a tendency to the development of aneurisms in various parts of the body. A remarkable instance of it in our profession was afforded by the brilliant Thomas King Chambers, who first had an aneurism in the left popliteal artery, eleven years subsequently an aneurism in the right leg which was cured by pressure, and finally aneurism of both carotid arteries.

Aneurism of the Thoracic Aorta.

The causes which favor the development of arterio-sclerosis prevail in aortic aneurism, particularly alcohol, syphilis, and overwork. The greatest danger probably is in strong muscular men with commencing degenerative processes in the arteries (a consequence of syphilis or alcohol or a result of hereditary weakness of the arterial tissues), who during a sudden muscular exertion are liable to lacerate the media, the intima not yet being strengthened by compensatory thickening over a spot of mesarteritis. Aneurisms of the thoracic aorta vary greatly in size and shape. A majority of them are saccular. They may be small and situated just above the aortic ring. Others form large tumors which project externally and occupy a large portion of the upper thorax. Small sacs from the descending portion of the arch may compress the trachea or the bronchi. In the tho-

racic portion the sac may erode the vertebræ or grow into the pleural cavity and compress the lung. In some instances it grows through the ribs and appears in the back.

Symptoms.—The chief influence of an aneurism is manifested in what are known as pressure effects. In the absence of these the aneurisms attain a large size without producing symptoms or seriously interfering with the circulation. Indeed, a useful clinical subdivision as given by Bramwell is into three groups—aneurisms which are entirely latent and give no physical signs; aneurisms which present signs of intrathoracic pressure, but it is difficult or impossible to determine the nature of the lesion producing the pressure; and, lastly, aneurisms which produce distinct tumors with well-marked pressure symptoms and external signs. It is perhaps best to consider aneurisms of the aorta according to the situation of the tumor.

(*a*) *Aneurisms of the Ascending Portion of the Arch.*—When just above the sinuses of Valsalva they are often small and latent. The first symptom may be rupture, which usually takes place into the pericardium and causes instant death. Above the sinuses, along the convex border of the ascending part, aneurism frequently develops, and may grow to a large size, either passing out into the right pleura or forward, pointing at the second or third interspace, eroding the ribs and sternum, and producing large external tumors. In this situation the sac is liable indeed to compress the superior vena cava, causing engorgement of the vessels of the head and arm, sometimes compressing only the subclavian vein, and causing enlargement and œdema of the right arm. Perforation may take place into the superior vena cava, of which accident Pepper and Griffith have collected twenty-nine cases. Large aneurisms in this situation may cause much dislocation of the heart, pushing it down and to the left, and sometimes compressing the inferior vena cava, and causing swelling of the feet and ascites. The right recurrent laryngeal nerve is often involved in these tumors. Death commonly follows from rupture into the pleura, or into the superior cava; less commonly from rupture externally, sometimes from heart-failure.

(*b*) *Aneurisms of the Transverse Arch.*—These may grow forward, erode the sternum, and produce large tumors. More commonly they are small and produce no external tumor, but cause marked pressure signs in their growth backward toward the spine, involving the trachea and the œsophagus, producing cough, which is often of a paroxysmal character, and dysphagia. The left recurrent laryngeal is often involved in its course round the arch. A small aneurism from the lower or posterior wall of the arch may compress a bronchus, inducing bronchorrhœa, gradual bronchiectasy, and suppuration in the lung—a process which by no means infrequently causes death in aneurism, and a condition which at the Montreal General Hospital we were in the habit of terming aneurismal phthisis. Occasionally enormous aneurisms develop in this situa-

tion, and grow into both pleuræ, extending between the manubrium and the vertebræ, and may persist for years. The sac may be evident at the sternal notch. The innominate, less commonly the left carotid and subclavian, may be involved in the sac, and the radial or carotid pulse may be absent or retarded. Pressure on the sympathetic may at first cause dilatation and subsequently contraction of the pupil. Sometimes the thoracic duct is compressed.

(*c*) *Aneurisms of the Descending Portion.*—Pressure signs are not so marked. The pain is often intense, owing to erosion of the vertebræ. Dysphagia may occur. Compression of the lung or compression of certain bronchi may induce bronchiectasy, retention of secretions, and fever. A tumor may appear externally in the region of the scapula, and here attain an enormous size. Occasionally the aneurisms in this region are small and latent, and prove fatal by rupture into the œsophagus. I have reported a case of sudden death, in which the heart and arch of the aorta were normal and the stomach was distended with blood, which could not be accounted for until the œsophagus was slit open, when it was found that a small aneurism in the thoracic aorta, smaller than a walnut, had ruptured into the gullet. The sac may erode the vertebræ and open the spinal canal, producing compression of the cord. Death not infrequently occurs from rupture into the pleura.

Diagnosis and Physical Signs.—*Inspection.*—In many instances this is negative. On either side of the sternum there may be abnormal pulsation, due to dislocation of the heart or to deformity of the thorax. The aneurismal pulsation is usually above the level of the third rib and most commonly to the right of the sternum, either in the first or second interspace. It may be only a diffuse heaving impulse without any external tumor. Often the impulse is noticed only when the chest is looked at obliquely in a favorable light. When the innominate is involved the throbbing may pass into the neck or be apparent at the sternal notch. Posteriorly, when pulsation occurs, it is most commonly found in the left scapular region. An external tumor is present in many cases, projecting either through the upper part of the sternum or to the right, sometimes involving the sternum and costal cartilages on both sides, forming a tumor the size of a cocoa-nut or even larger. The skin is thin, often blood-stained, or it may have ruptured, exposing the laminæ of the sac. The apex beat may be much dislocated, particularly when the sac is large. It is more commonly a dislocation from pressure than from enlargement of the heart itself.

Palpation.—The area and degree of pulsation are best determined by palpation. When the aneurism is deep-seated and not apparent externally, the bimanual method should be used, one hand upon the spine and the other on the sternum. When the sac has perforated the chest wall the impulse is, as a rule, forcible, slow, heaving, and expansile. The resistance may be very great if there are thick laminæ beneath the skin;

more rarely the sac is soft and fluctuating. The hand upon the sac, or on the region in which it is in contact with the chest wall, feels in many cases a diastolic shock, often of great intensity, which forms one of the valuable physical signs of aneurism. A systolic thrill is sometimes present, not so often in saccular aneurisms as in the dilatation of the arch. The pulsation may sometimes be felt in the suprasternal notch.

Percussion.—The small and deep-seated aneurisms are in this respect negative. In the larger tumors, as soon as the sac reaches the chest wall, there is produced an area of abnormal dulness, the position of which depends upon the part of the aorta affected. Aneurisms of the ascending arch grow forward and to the right, producing dulness on one side of the manubrium; those from the transverse arch produce dulness in the middle line, extending toward the left of the sternum, while aneurisms of the descending portion most commonly produce dulness in the left interscapular and scapular regions. The percussion note is flat and gives a feeling of increased resistance.

Auscultation.—Adventitious sounds are not always to be heard. Even in a large sac there may be no murmur. Much depends upon the thickness of the laminæ of fibrin. An important sign, particularly if heard over a dull region, is a ringing, accentuated second sound, a phenomenon rarely missed in large aneurisms of the aortic arch. A systolic murmur may be present; sometimes a double murmur, in which case the diastolic *bruit* is usually due to associated aortic insufficiency. The systolic murmur alone is of little moment in the diagnosis of an aneurismal sac. With the single stethoscope the shock of the impulse with the first sound is sometimes very marked.

Among other physical signs of importance are slowing of the pulse in the arteries beyond the aneurism, or in those involved in the sac. There may, for instance, be a marked difference between the right and left radial, both in volume and time. A physical sign of large thoracic aneurism which I have not seen referred to is obliteration of the pulse in the abdominal aorta and its branches. My attention was called to this in a patient who was stated to have aortic insufficiency. There was a well-marked diastolic murmur, but in the femorals and in the aorta I was surprised to find no trace of pulsation, and not the slightest throbbing in the abdominal aorta or in the peripheral arteries of the leg. The circulation was, however, unimpaired in them and there was no dilatation of the veins. Attracted by this, I then made a careful examination of the patient's back, when the circumstance was discovered, which neither the patient himself nor any of his physicians had noticed, that he had a very large area of pulsation in the left scapular region. The sac probably was large enough to act as a reservoir annihilating the ventricular systole, and converting the intermittent into a continuous stream.

The *tracheal tugging*, a valuable sign in deep-seated aneurisms, was described by Surgeon-Major Porter, and has been specially studied by my

colleagues Ross and MacDonnell * at the Montreal General Hospital. To test it the patient should sit up with the head inclined forward, so as completely to relax the neck. The cricoid cartilage is grasped between the index-finger and the thumb and by upward pressure the trachea put upon the stretch. In healthy individuals no sensation is felt, but if an aneurism is attached to it or is adherent in the immediate vicinity, the stretching is accompanied by a well-marked and characteristic tugging. On several occasions I have known this to be a sign of great value in the diagnosis of deep-seated aneurisms. I have never felt it in tumors, or in the extreme dynamic dilatation of aortic insufficiency.

Occasionally a systolic murmur may be heard in the trachea, as pointed out by David Drummond, or even at the patient's mouth, when opened. This is either the sound conveyed from the sac, or is produced by the air as it is driven out of the wind-pipe during the systole.

An important but variable feature in thoracic aneurism is *pain*, which is particularly marked in deep-seated tumors. It is usually paroxysmal, sharp, and lancinating, often very severe when the tumor is eroding the vertebræ, or perforating the chest wall. In the latter case, after perforation the pain may cease. Anginal attacks are not uncommon, particularly in aneurisms at the root of the aorta. Frequently the pain radiates down the left arm or up the neck, sometimes along the upper intercostal nerves. *Cough* results either from the direct pressure on the wind-pipe, or is associated with bronchitis. The expectoration in these instances is abundant, thin, and watery; subsequently it becomes thick and turbid. Paroxysmal cough of a peculiar brazen, ringing character is a characteristic symptom in some cases, particularly when there is pressure on the recurrent laryngeal nerves.

Dyspnœa, which is common in cases of aneurism of the transverse portion, is not necessarily associated with pressure on the recurrent laryngeal nerves, but may be due directly to compression of the trachea or the left bronchus. It may occur with marked stridor. Loss of voice and hoarseness are consequences of pressure on the recurrent laryngeal, usually the left, inducing either a spasm in the muscles of the left vocal cord or paralysis.

Paralysis of an abductor on one side may be present without any symptoms. It is more particularly, as Semon states, when the paralytic contractures supervene that the attention is called to laryngeal symptoms.

Hæmorrhage in thoracic aneurism may come from (*a*) the soft granulations in the trachea at the point of compression, in which case the sputa are blood-tinged, but large quantities of blood are not lost; (*b*) from rupture of the sac into the trachea or bronchi; (*c*) from perforation into the lung or erosion of the lung tissue. The bleeding may be profuse, rapidly proving fatal, and is a common cause of death. It may persist for weeks or

* London Lancet, 1891.

months, in which case it is simply hæmorrhagic weeping through the sac, which is exposed in the trachea. In some instances, even after a very profuse hæmorrhage, the patient recovers and may live for years. A patient with well-marked thoracic aneurism, whom I showed to my class at the University of Pennsylvania four years ago and who had had several brisk hæmorrhages, died recently, having in the mean time enjoyed average health.

Difficulty of swallowing is a comparatively rare symptom, and may be due either to spasm or to direct compression. The sound should never be passed in these cases, as the œsophagus may be almost eroded and a perforation may be made.

Among other signs and symptoms venous compression, which has already been mentioned, may involve one subclavian or the superior vena cava. A curious phenomenon in intrathoracic aneurism is the clubbing of the fingers and incurving of the nails of one hand, of which two examples have been under my care, in both without any special distention or signs of venous engorgement. Tumors of the arch may involve the pulmonary artery, producing compression, or in some instances adhesion of the pulmonary segments and insufficiency of the valve; or the sac may rupture into the artery, an accident which happened in two of my cases, producing instantaneous death.

Pressure on the sympathetic is particularly liable to occur in growths from the ascending portion of the arch. Either the upper dorsal or the lower cervical ganglion is involved. The symptoms are variable. If the nerve is simply irritated there is stimulation of the vaso-dilator fibres and dilatation of the pupil. With this may be associated pallor of the same side of the face. On the other hand, destruction of the cilio-spinal branches causes paralysis of the dilator fibres, in consequence of which the iris contracts, the vessels on the side of the head dilate, causing congestion, and in some instances unilateral sweating. It is much more common to see the pupillary symptoms alone than in combination either with pallor, redness, or sweating.

The clinical picture of aneurism of the aorta is extremely varied. Many cases present characteristic symptoms and no physical signs, while others have well-marked physical signs and no symptoms. As Broadbent remarks, the aneurism of *physical signs* springs from the ascending portion of the aorta; the aneurism of *symptoms* grows from the transverse arch.

Aneurism of the aorta may be confounded with: (*a*) The violent throbbing impulse of the arch in aortic insufficiency. I have already referred to a case of this kind in which the diagnosis of aneurism was made by several good observers. In a case recently under observation dulness and pulsation existed in the second right interspace with a well-marked systolic and a loud diastolic murmur, which was heard far out in the right mammary region. The question arose whether aneurism was present in

addition to the aortic insufficiency. The post-mortem showed the margin of the right lung retracted and adherent to the pericardium, leaving exposed the aorta, which must have been greatly distended during each systole.

(*b*) *Simple Dynamic Pulsation.*—No instance of this, which is common in the abdominal aorta, has ever come under my notice. One which came under the care of William Murray and Bramwell presented, without any pain or pressure symptoms, pulsation and dulness over the aorta. The condition gradually disappeared and was thought to be neurotic.

(*c*) Dislocation of the heart in curvature of the spine may cause great displacement of the aorta, so that it has been known to pulsate forcibly to the right of the sternum.

(*d*) *Solid Tumors.*—When the tumor projects externally and pulsates the difficulty may be considerable. In tumor the heaving, *expansile* pulsation is absent, and there is not that sense of force and power which is so striking in the throbbing of a perforated aneurism. There is not to be felt as in aortic aneurism the shock of the heart-sounds, particularly the diastolic shock. Auscultatory sounds are less definite, as large aneurisms may occur without murmur; and, on the other hand, murmurs may be heard over tumors. The greatest difficulty is in the deep-seated thoracic tumors, and here the diagnosis may be impossible. I have already referred to the case which was regarded by Skoda as aneurism and by Oppolzer as tumor. The physical signs may be indefinite. The ringing aortic second sound is of great importance and is rarely, if ever, heard over tumor. Tracheal tugging is here a valuable sign. Pressure phenomena are less common in tumor, whereas pain is more frequent. The general appearance of the patient in aneurism is much better than in tumor. There may be signs of enlargement of the glands in the axilla or in the neck. Healthy, strong males who have worked hard and have had syphilis are the most common subjects of aneurism. Occasionally cancer of the œsophagus may simulate aneurism, producing pressure on the left bronchus, and in one instance at the Philadelphia Hospital, with a husky, brazen cough, the symptoms were very suggestive.

(*e*) *Pulsating Pleurisy.*—In cases of *empyema necessitatis*, if the projecting tumor is in the neighborhood of the heart and pulsates, the condition may readily be mistaken for aneurism. The absence of the heaving, firm distention and of the diastolic shock would, together with the history and the existence of pleural effusion, determine the nature of the case. If necessary, puncture may be made with a fine hypodermic needle. In a majority of the cases of pulsating pleurisy the throbbing is diffuse and widespread, moving the whole side.

Prognosis.—The outlook in thoracic aneurism is always grave. Life may be prolonged for some years, but the patients are in constant jeopardy. Spontaneous cure is not very infrequent in the small sacculated tumors of the ascending and thoracic portions. The cavity becomes filled with lam-

inæ of firm fibrin, which become more and more dense and hard, the sac shrinks considerably, and finally lime salts are deposited in the old fibrin. The laminæ of fibrin may be on a level with the lumen of the vessel, causing complete obliteration of the sac. The cases which rupture externally, as a rule run a rapid course, although to this there are exceptions; the sac may contract, become firm and hard, and the patient may live for five, or even, as in a case mentioned by Balfour, for ten years. The cases which have lasted longest in my experience have been those in which a saccular aneurism has projected from the ascending arch. One patient in Montreal had been known to have aneurism for eleven years. The aneurism may be enormous, occupying a large area of the chest, and yet life be prolonged for many years, as in the case mentioned as under the care of Skoda and Oppolzer. One of the most remarkable instances is the case of dissecting aneurism reported by Graham. The patient was invalided after the Crimean War with aneurism of the aorta, and for years was under the observation of J. H. Richardson, of Toronto, under whose care he died in 1885. The autopsy showed a healed aneurism of the arch, with a dissecting aneurism passing the whole length of the aorta, which formed a double tube.

Treatment.—In a large proportion of the cases this can only be palliative. Still in every case measures should be taken which are known to promote clotting and consolidation within the sac. In any large series of cured aneurisms a considerable majority of the patients have not been known to be subjects of the disease, but the obliterated sac has been found accidentally at the post mortem.

The most satisfactory plan in early cases, when it can be carried out thoroughly, is that advised by the late Mr. Tufnell, of Dublin, the essentials of which are rest and a restricted diet. Rest is essential and should, as far as possible, be absolute. The reduction of the daily number of heart-beats when a patient is recumbent and makes no exertion whatever amounts to many thousands, and is one of the principal advantages of this plan. Mental quiet should also be enjoined. The diet advised by Tufnell is extremely rigid—for breakfast, two ounces of bread and butter and two ounces of milk; for dinner, two or three ounces of meat and three or four ounces of milk or claret; for supper, two ounces of bread and two ounces of milk. This low diet diminishes the blood-volume and is thought also to render the blood more fibrinous. It reduces greatly the blood-pressure within the sac, in this manner favoring coagulation. This treatment should be pursued for several months, but, except in persons of a good deal of mental stamina, it is impossible to carry it out for more than a few weeks at a time. It is a form of treatment adapted only for the saccular form of aneurism, and in cases of large sacs communicating with the aorta by a comparatively small orifice the chances of consolidation are fairly good. Unquestionably rest and the restriction of the liquids are the important parts of the treatment, and a greater

variety and quantity of food may be allowed with advantage. If this plan cannot be thoroughly carried out, the patient should at any rate be advised to live a very quiet life, moving about with deliberation and avoiding all sudden mental or bodily excitement. The bowels should be kept regular, and constipation and straining should be carefully avoided. Of medicines, iodide of potassium, as advised by Balfour, is of great value. It may be given in doses of from ten to fifteen or twenty grains three times a day. Larger doses are not necessary. The mode of action is not well understood. It may act by increasing the secretions and so inspissating the blood, by lowering the blood-pressure, or, as Balfour thinks, by causing thickening and contraction of the sac. The most striking effect of the iodide in my experience has been the relief of the pain. The evidence is not conclusive that the syphilitic cases are more benefited than the non-syphilitic. All these measures have little value unless the sac is of a suitable form and size. The large tumors with wide mouths communicating with the ascending portion of the aorta may be treated on the most approved plans for months without the slightest influence other than reduction in the intensity of the throbbing. A patient with a tumor projecting into the right pleura remained on the most rigid Tufnell treatment for more than one hundred days, during which time he also took iodide of potassium faithfully. The pulsations were greatly reduced and the area of dulness diminished, and we congratulated ourselves that the sac was probably consolidating. Sudden death followed rupture into the pleura, and the sac contained only fluid blood, not a shred of fibrin. In cases in which the tumor is large, or in which there seems to be very little prospect of consolidation, it is perhaps better to advise a man to go on quietly with his occupation, avoiding excitement and worry. Our profession has offered many examples of good work thoroughly and conscientiously carried out by men with aneurism of the aorta, who wisely, I think, as the late Hilton Fagge, preferred to die in harness. Other measures to induce coagulation in the sac are electricity, which has occasionally proved successful; the insertion of horse-hair, thin wire, or needles; the injection of an astringent liquid, such as perchloride of iron, into the sac. In a few cases only these have been followed by cure. The fine silver wire pushed through a hypodermic needle is probably the most satisfactory method, and may be combined with electrolysis, the method known as Loreta's. Kerr and Rosenstein, of San Francisco, have recently reported cases in which cure was effected in this way.

Other Symptoms requiring Treatment.—Pressure on veins causing engorgement, particularly of the head and arms, is sometimes promptly relieved by free venesection, and at any time during the course of a thoracic aneurism, if attacks of dyspnœa with lividity supervene, bleeding may be resorted to with great benefit. It has the advantage also of promptly checking the pain, for which symptom, as already mentioned, the iodide of potassium often gives relief. In the final stages morphia is, as a

rule, necessary. Dyspnœa, if associated with cyanosis, is best relieved by bleeding. Chloroform inhalations may be necessary. The question sometimes comes up with reference to tracheotomy in these cases of urgent dyspnœa. If it can be shown by laryngoscopic examination that it is due to bilateral abductor paralysis the trachea may be opened, but this is extremely rare, and in nearly every instance the urgent dyspnœa is caused by pressure about the bifurcation. When the sac appears externally and grows large an ice-cap may be applied upon it, or a belladonna plaster to allay the pain. In some instances an elastic support may be used with advantage, and I saw a physician with an enormous external aneurism in the right mammary region who for many months had obtained great relief by the elastic support, passing over the shoulder and under the arm of the opposite side.

Digitalis, ergot, aconite, and veratrum viride are rarely, if ever, of service in thoracic aneurism.

Aneurism of the Abdominal Aorta.

The sac is most common in the neighborhood of the cœliac axis. It is rare in comparison with thoracic aneurism. The tumor may be fusiform or sacculated, and it is sometimes multiple. Projecting backward, it erodes the vertebræ and may cause numbness and tingling in the legs and finally paraplegia, or it may pass into the thorax and burst into the pleura. More commonly the sac is on the anterior wall and projects forward as a definite tumor, which may be either in the middle line or a little to the left. The tumor may be large and evident, or when high up beneath the pillar of the diaphragm it may attain considerable size without being very apparent on palpation.

The symptoms are chiefly pain, very often of a cardialgic nature, passing round the sides or localized in the back, and gastric symptoms, particularly vomiting. Retardation of the pulse in the femoral is a very common symptom.

Diagnosis and Physical Signs.—Inspection may show marked pulsation in the epigastric region, sometimes a definite tumor. A thrill is not uncommon. The pulsation is forcible, expansile, and sometimes double when the sac is large and in contact with the pericardium. On palpation a *definite tumor can be felt.* If large, there is some degree of dulness on percussion which usually merges with that of the left lobe of the liver. On auscultation, a systolic murmur is, as a rule, audible, and is sometimes best heard at the back. A diastolic murmur is occasionally present, usually very soft in quality. One of the commonest of clinical errors is to mistake a throbbing aorta for an aneurism. It is to be remembered that no pulsation, however forcible, or the presence of a thrill or a systolic murmur justifies the diagnosis of abdominal aneurism unless there is a *definite tumor which can be grasped and which has an expansile pulsa-*

tion. Attention to this rule will save many errors. The throbbing or pulsating aorta is met with in all neurasthenic conditions, particularly in women, and it is remarkable with what violence the epigastrium may be driven out with each systole. In anæmia, particularly some instances of traumatic anæmia, the throbbing may be very great. In the case of a large, stout man with severe hæmorrhages from a duodenal ulcer the throbbing of the abdominal aorta not only shook violently the whole abdomen, but communicated a pulsation to the bed, the shock of which was distinctly perceptible to any one sitting upon it. Very frequently a tumor of the pylorus, of the pancreas, or of the left lobe of the liver is lifted with each impulse of the aorta and may be confounded with aneurism. The absence of the forcible expansile impulse and the examination in the knee-elbow position, in which the tumor, as a rule, falls forward, and the pulsation is not then communicated, suffice for differentiation.

The outlook in abdominal aneurism is bad. A few cases heal spontaneously. Death may result from (*a*) complete obliteration of the lumen by clots; (*b*) compression paraplegia; (*c*) rupture either into the pleura, retroperitoneal tissues, peritonæum or the intestines, very commonly the duodenum; (*d*) by embolism of the superior mesenteric artery, producing infarction of the intestines.

The *treatment* is such as already advised in thoracic aneurism. When the aneurism is low down pressure has been successfully applied in a case by Murray, of Newcastle. It must be kept up for many hours under chloroform. The plan is not without risk, as patients have died from bruising and injury of the sac.

Aneurism of the Branches of the Abdominal Aorta.

The *cœliac axis* is itself not infrequently involved in aneurism of the first portion of the abdominal aorta. Of its branches, the *splenic artery* is occasionally the seat of aneurism. This rarely causes tumor large enough to be felt; sometimes, however, the tumor is of large size. I have reported the case of a man, aged thirty, who had an illness of several months' duration, severe epigastric pain and vomiting, which led his physicians in New York to diagnose gastric ulcer. There was a deep-seated tumor in the left hypochondriac region, the dulness of which merged with that of the spleen. There was no pulsation, but it was thought on one occasion that a *bruit* was heard. The chief symptoms while under observation were vomiting, severe epigastric pain, occasional hæmatemesis, and finally severe hæmorrhage from the bowels. An aneurism of the splenic artery the size of a cocoa-nut was situated between the stomach above and the transverse colon below, and extended to the left as far as the level of the navel. The sac contained densely laminated fibrin. It had perforated the colon. I have twice seen small aneurisms on the splenic artery. Of thirty-nine instances

of aneurism on the branches of the abdominal aorta collected by Lebert, ten were of the splenic artery.

Aneurism of the *hepatic artery* is very rare, and there are only ten or twelve cases on record. The symptoms are extremely indefinite; the condition could rarely be diagnosed. In the case reported by Ross and myself, a man aged twenty-one had the symptoms of pyæmia. The liver was greatly enlarged, weighed nearly 5,000 grammes, and presented innumerable small abscesses. An oval aneurism, half the size of a small lemon, involved the right and part of the left branches.

A few cases of aneurism of the *superior mesenteric artery* are on record. The diagnosis is scarcely possible. Plugging of the branches or of the main stem may cause the symptoms of infarction of the bowels which have already been considered.

Small aneurisms of the *renal artery* are not very uncommon. Large tumors are rare. The sac may rupture and give rise to extensive retroperitoneal hæmorrhage.

Arterio-venous Aneurism.

In this form there is abnormal communication between an artery and a vein. When a tumor lies between the two it is known as varicose aneurism; when there is a direct communication without tumor the vein is chiefly distended and the condition is known as aneurismal varix.

An aneurism of the ascending portion of the arch may open directly into the vena cava. Twenty-nine cases of this lesion have been analyzed by Pepper and Griffith. Cyanosis, œdema, and great distention of the veins of the upper part of the body are the most frequent symptoms, and develop, as a rule, with suddenness. Of the physical signs a thrill is present in some cases. A continuous murmur with systolic intensification is of great diagnostic value. In a recent case, after the existence for some time of pressure symptoms, intense cyanosis developed with engorgement of the veins of the head and arms. Over the aortic region there was a loud continuous murmur with systolic intensification.

A majority of the cases of arterio-venous aneurism and of aneurismal varix result from the accidental opening of an artery and vein as in venesection, and are met with at the bend of the elbow or sometimes in the temporal region. The condition may persist for years without causing any trouble. Pulsation, a loud thrill, and a continuous humming murmur are usually present.

Congenital Aneurism.

In consequence of failure of proper development of the elastic coat in many places in the arterial system, multiple aneurisms may develop. In the well-known case described by Küssmaul and Maier, upon many of the

medium-sized arteries there were nodular prominences, which consisted of thickening of the intima and infiltration of the adventitia and of the media, with a nuclear growth which in places looked quite sarcomatous. They called it a case of *periarteritis nodosa*, and Eppinger holds that it belongs to the category which he makes of congenital aneurism. As many as sixty-three aneurismal tumors have been found in one case. In the smaller branches, such as the coronary and the mesenteric arteries or in the pulmonary arteries, there may be numerous elongated or saccular aneurisms varying in size from a cherry to a hazel-nut. These are true aneurismal dilatations, and, according to Eppinger's careful study, consist of the intima and the adventitia, the elastic lamina having disappeared. The condition has been met with in children Some of the cases, however, have been in adults; but the term as applied by Eppinger expresses, and probably correctly, the deep-seated fundamental error in development which must be at the basis of this condition. The coronary arteries is a favorite situation; a case has been reported by Gee in a boy of seven.

SECTION VI.

DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

I. ANÆMIA.

ANÆMIA may be defined as a reduction in the amount of the blood as a whole or of its corpuscles, or of certain of its more important constituents, such as albumen and hæmoglobin. The condition may be general or local. The former alone we are here considering. It is interesting to note, however, that the pallor, particularly of the face, which is one of the most striking symptoms of anæmia, is just as characteristic of local anæmia due to fright or to nausea. There are persons persistently pale without actual anæmia in whom the condition may be due to inherited peculiarities.

Our knowledge is not yet sufficiently advanced to classify satisfactorily the various forms of anæmia. The following provisional grouping may be made: (1) Secondary or symptomatic anæmia; (2) primary, essential, or cytogenic anæmia.

SECONDARY ANÆMIA.

Under this division comes a large proportion of all cases. The following are the most important groups, based on the etiology:

(1) *Anæmia from hæmorrhage*, either traumatic or spontaneous. The loss of blood may be rapid, as in lesions of large vessels, in injury or in rupture of aneurisms, or in cases of ulcer of the stomach or duodenum, or post-partum hæmorrhage. If the loss is excessive, death results from lowering of the arterial pressure. In sudden profuse hæmorrhage the loss of three or four pounds of blood may prove fatal. In the rupture of an aneurism into the pleura the loss of blood may amount to seven pounds and a half, the largest quantity I have known to be shed into one cavity. In a case of hæmatemesis the patient lost over ten pounds by measurement in one week and yet recovered from the immediate effects. Even after very severe hæmorrhage the number of red blood-corpuscles is not reduced so greatly as in forms of idiopathic anæmia. Thus in a case just

mentioned, at the termination of the week of bleeding there were nearly 1,390,000 red blood-corpuscles to the cubic millimetre. The process of regeneration goes on with great rapidity, and in some "bleeders" a week or ten days suffice to re-establish the normal amount. The watery and saline constituents of the blood are readily restored by absorption from the gastro-intestinal tract. The albuminous elements also are quickly renewed, but it may take weeks or months for the corpuscles to reach the

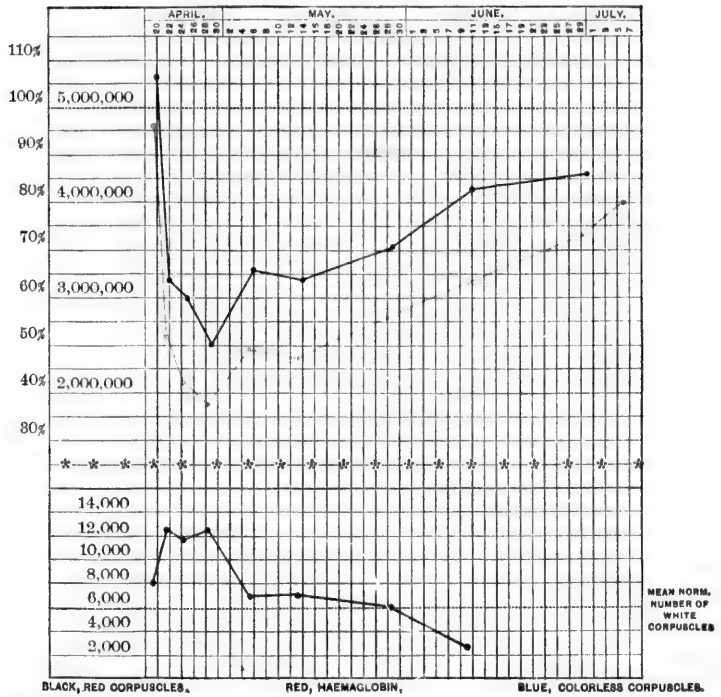


CHART XVI.—Illustrates the rapidity with which anæmia is produced in purpura hæmorrhagica and the gradual recovery.*

normal standard. The accompanying chart illustrates the rapid fall and gradual restitution in a case of severe purpura hæmorrhagica.

The microscopical characters of the blood after severe hæmorrhage are not much changed; the white corpuscles are relatively increased, producing a condition of leucocytosis. Nucleated red corpuscles are present, though usually not numerous. In the regeneration of the blood the de-

* On September 27th the patient returned from the country, where she had spent the summer. The blood count was then: Red corpuscles, 5,350,000; white corpuscles, 5,500; hæmoglobin, ninety-four per cent.

velopment of the hæmoglobin does not keep pace with that of the corpuscles.

(2) Anæmia is frequently produced by long-continued drain on the albuminous materials of the blood, as in chronic suppuration and Bright's disease. Prolonged lactation acts in the same way. Rapidly growing tumors may cause a profound anæmia, as in gastric cancer. The character of the blood in these cases is similar to that in anæmia after hæmorrhage.

(3) *Anæmia from Inanition.*—This may be brought about by defective food supply, or by conditions which interfere with the proper reception and preparation of the food, as in cancer of the œsophagus and chronic dyspepsia. The reduction of the blood mass may be extreme, but the plasma suffers proportionately more than the corpuscles, which, even in the wasting of cancer of the œsophagus, may not be reduced more than one half or three fourths.

(4) *Toxic anæmia*, induced by the action of certain poisons on the blood, such as lead, mercury, and arsenic, among inorganic substances, and the virus of syphilis and malaria among organic poisons. They act either by directly destroying the red blood-corpuscles, as in malaria, or by increasing the rate of ordinary consumption. The anæmia of pyrexia may in part be due to a toxic action, but is also caused in part by the disturbance of digestion and interference with the function of the blood-making organs.

Primary or Essential Anæmia.

1. **Chlorosis.**—An essential anæmiæ met with chiefly in young girls, characterized by a marked relative diminution of the hæmoglobin.

Etiology.—Cases are rarely seen in men. Blondes are more frequently affected than brunettes. The age of onset is usually between the fourteenth and the seventeenth years. Recurrences throughout the third decade are, however, not uncommon. Chlorósis is extremely rare in young children.

Hereditary influences probably play a part. Virchow pointed out that in many cases there was a defective development of the circulatory system, either congenital or resulting in a failure of the normal rate of growth. In some instances a compensatory hypertrophy of the heart has been found.

The disease is most common among the ill-fed, overworked girls of large towns, who are confined all day in close, badly-lighted rooms, or have to do much stair-climbing. Cases are frequent, however, under the most favorable conditions of life. Lack of proper exercise and fresh air, and improper food are important factors. Emotional and nervous disturbances may be prominent, so prominent that certain writers have regarded the disease as a neurosis. Menstrual disturbances are not un-

common, but are probably a sequence, not a cause of chlorosis. Sir Andrew Clark believes that constipation plays an important *rôle*, and that the condition is in reality a *copræmia* due to the absorption of poisons—leucomaines and ptomaines—from the large bowel.

Morbid Anatomy.—Fortunately the disease is rarely fatal. The fat is well retained. Hypoplasia of the aorta and larger arteries has been found in some cases, and the vessels have had a remarkable degree of elasticity. The heart is usually dilated and the left ventricle hypertrophied. Hypoplasia of the uterus and defective development of the genitalia have also been found.

Symptoms.—The blood examination: Johann Duncan in 1867 first called attention to the fact that the essential feature was not a quantitative but a qualitative change in the hæmoglobin. This has been abundantly confirmed. The red blood-corpuscles may show only a moderate grade of reduction, but the corpuscles themselves are very poor in hæmoglobin. Thus in forty consecutive cases examined at my clinic by Thayer, the average number per cubic millimetre of the red blood-corpuscles was 4,225,181, or over eighty per cent, whereas the percentage of hæmoglobin for the total number was 44·1 per cent. The accompanying chart illustrates well these striking differences. The least blood-count in the series of cases referred to above was 1,932,000. There may be all the physical characteristics and symptoms of a profound anæmia with blood-corpuscles nearly at the normal standard. Thus in one instance the globular richness was over eighty-five per cent with the hæmoglobin about thirty-five. These characteristics are distinctive, I believe, and not found in the same grade in any other form of anæmia. The importance of the reduction in the hæmoglobin depends upon the fact that it is the iron-containing element of the blood with which in respiration the oxygen enters into combination. This marked diminution in the iron has also been determined by chemical analysis of the blood. The microscopical characteristics of the blood are as follows: In severe cases the corpuscles may be extremely irregular in size—poikilocytosis—which may occasionally be as marked as in pernicious anæmia. The large forms of red blood-cells are common, but the average size is stated to be below normal. The color of the corpuscles is noticeably pale and the deficiency may be seen either in individual corpuscles or in the blood mixture prepared for counting. The leucocytes may show a slight increase; thus, the average of the forty cases above referred to was 8,256 per cubic millimetre.

The general symptoms of chlorosis are those of an anæmia of moderate grade. The subcutaneous fat is well retained or even increased in amount. The complexion is peculiar; neither the blanched aspect of hæmorrhage nor the muddy pallor of grave anæmia, but a curious yellow-green tinge which has given the name to the disease, and to its popular designation, the green sickness. In cases of moderate grade the color may be deceptive, as the cheeks have a reddish tint, particularly on exertion (chlorosis

rubra). The subjects complain of breathlessness and palpitation, and there may be a tendency to fainting. The palpitation and breathlessness

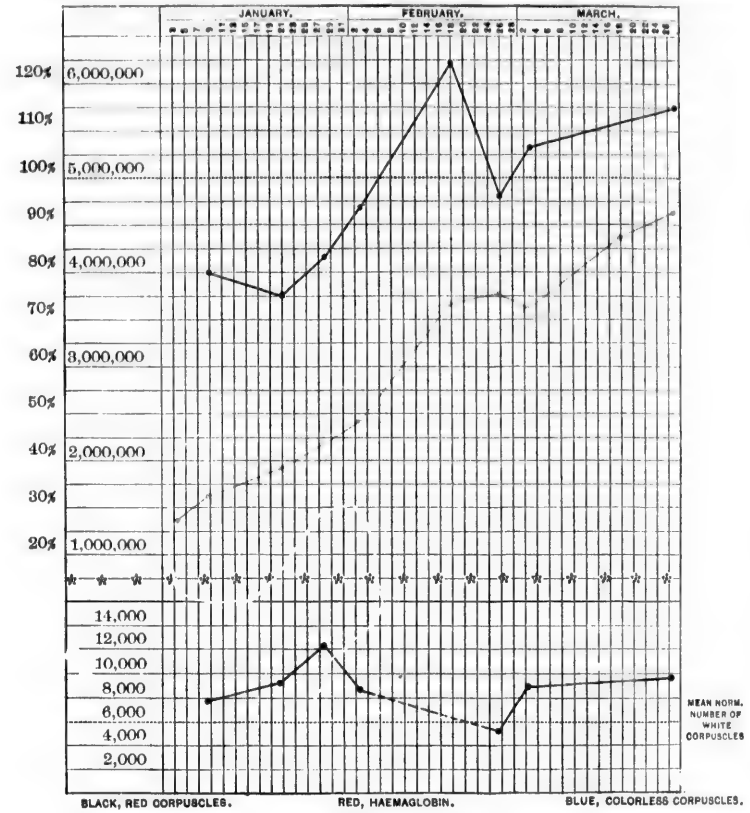


CHART XVII.—Chlorosis,

often lead to the suspicion of heart or lung disease. The eyes have a peculiar brilliancy and the sclerotics are of a sky-blue color Occasionally the skin shows areas of pigmentation, particularly about the joints.

Digestive symptoms are common. The appetite is capricious and the patients often have a longing for unusual articles, particularly acids. In some instances they eat all sorts of indigestible things, such as chalk or even earth. Constipation is a common symptom, and, as already mentioned, has been regarded as an important element in causing the disease. Contourier has noted the frequent association of dilatation of the stomach with chlorosis, and states that in some cases this may be an etiological factor, while in others it may be a result.

The circulatory symptoms are important. Palpitation of the heart oc-

curs on exertion, and may be the most distressing symptom of which the patient complains. Percussion may show slight increase in the transverse dulness. A systolic murmur is heard at the apex or at the base; more commonly at the latter, but in extreme cases at both. A diastolic murmur is rarely heard. The systolic murmur is usually loudest in the second left intercostal space, where there is sometimes a distinct pulsation. The exact mode of production is still in dispute. Balfour holds that it is produced at the mitral orifice by relative insufficiency of the valves in the dilated condition of the ventricle. On the right side of the neck over the jugular vein a continuous murmur is heard, the *bruit de diable*, or humming-top murmur.

The pulse is usually full and soft. Pulsation in the peripheral veins is sometimes seen. There is a tendency to thrombosis in the veins; most commonly in the femoral, but in other instances in the longitudinal sinus, or the thrombosis may be multiple. Except in the sinuses, the condition is rarely serious. Tuckwell has reported an instance in which there was embolism of the right axillary artery with the loss of a thumb and part of the fingers. Brayton Ball has recently called attention to the importance of this feature of chlorosis.

As in all forms of essential anæmia, fever is not uncommon. Especial attention has of late been directed to this by French writers.* Chlorotic patients suffer frequently from headache and neuralgia, which may be paroxysmal. Hysterical manifestations are not infrequent. Menstrual disturbances are very common—amenorrhœa or dysmenorrhœa. With the improvement in the blood condition this function is usually restored.

Diagnosis.—The green sickness, as it is sometimes called, is in many instances recognized at a glance. The well-nourished condition of the girl, the peculiar complexion, which is most marked in brunettes, and the white sclerotics are very characteristic. A special danger exists in mistaking the anæmia of the early stage of pulmonary tuberculosis for chlorosis. The palpitation of the heart and shortness of breath frequently suggest heart-disease, and the œdema of the feet and general pallor cause the cases to be mistaken for Bright's disease. In the great majority of cases the characters of the blood readily separate chlorosis from other forms of anæmia.

2. **Idiopathic or Progressive Pernicious Anæmia.**—The disease was first clearly described by Addison, who called it idiopathic anæmia. Channing and Gusserow described the cases occurring post partum, but to Biermer we owe a revival of interest in the subject.

Etiology.—The existence of a separate disease worthy of the term progressive pernicious anæmia has been doubted, but there are unquestionably cases in which, as Addison says, there exist none of the usual causes or concomitants of anæmia. Clinically there are several different

* Trazit, Paris Thesis, 1888.

groups which present the characters of a progressive and pernicious anæmia and are etiologically different. Thus, a fatal anæmia may be due to the presence of parasites, or may follow hæmorrhage, or be associated with chronic atrophy of the stomach; but when we have excluded all these causes there remains a group which, in the words of Addison, is characterized by a "general anæmia occurring without any discoverable cause whatever, cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease."

Idiopathic anæmia is widely distributed. It is of frequent occurrence in the Swiss Cantons, and is not uncommon in this country. It affects middle-aged persons, but instances in children have been described. Griffith mentions about ten cases occurring under twelve years of age. The youngest patient I have seen was a girl of twenty. Males are more frequently affected than females. Of my 27 cases, 10 were females and 17 were males. Of 110 cases collected by Coupland, 56 were in men and 54 in women.

With the following conditions may be associated a profound anæmia not to be distinguished clinically from Addison's idiopathic form:

(*a*) *Pregnancy and Parturition.*—The symptoms may develop during pregnancy, as in 19 of 29 cases of this group in Eichhorst's table. More commonly, in my experience, the condition has been post partum; thus, of my 27 cases, 5 followed delivery.

(*b*) *Atrophy of the Stomach.*—This condition, early recognized by Flint and Fenwick, may certainly cause a progressive pernicious anæmia. By modern methods it may now be possible to exclude this extreme gastric atrophy.

(*c*) *Parasites.*—The most severe form may be due to the presence of parasites, and the accounts of cases depending upon the anchylostoma and the bothriocephalus describe a progressive and often pernicious anæmia.

After the exclusion of these forms there remains a large proportion, numbering eighteen cases in my series, which correspond to Addison's description. The etiology of these cases is still dark. The researches of Quincke and his student Peters showed that there was an enormous increase in the iron in the liver, and he suggested that the affection was probably due to increased hæmolysis. This has been strongly supported by the extensive observations of Hunter, who has also shown that the urine excreted is darker in color and contains pathological urobilin. The lemon tint of the skin or the actual jaundice is attributed, on this view, to the changes in the liver cells produced by the excessive amount of pigment, but in the light grades it is unquestionably hæmatogenous. To explain the hæmolysis, it has been thought that in the condition of faulty gastro-intestinal digestion, which is so commonly associated with these cases, poisonous materials are developed, which when absorbed cause destruction of the corpuscles. Certainly the evidence for hæmolysis is very

strong, but we are still far away from a full knowledge of the conditions under which it is produced.

On the other hand, F. P. Henry, Stephen Mackenzie, and other authorities incline to the belief that the essence of the disease is in defective hæmogenesis, in consequence of which the red blood-corpuscles are abnormally vulnerable. A point noted by Copeman, that the hæmoglobin crystallizes from the blood-corpuscles with great readiness, can scarcely be regarded as favoring the view of imperfect hæmogenesis, since this is a feature specially characteristic of the blood of the young.

Morbid Anatomy.—The body is rarely emaciated. A lemon tint of the skin is present in a majority of the cases. The muscles often are intensely red in color, like horse-flesh, while the fat is light yellow. Hæmorrhages are common on the skin and serous surfaces. The heart is usually large, flabby, and empty. In one instance I obtained only two drachms of blood from the right heart, and between three and four from the left. The muscle substance of the heart is intensely fatty, and of a pale, light-yellow color. In no affection do we see more extreme fatty degeneration. The lungs show no special changes. The stomach in many instances is normal, but in some cases of fatal anæmia the mucosa has been extensively atrophied. In the case described by Henry and myself the mucous membrane had a smooth, cuticular appearance, and there was complete atrophy of the secreting tubules. The liver may be enlarged and fatty. In most of my autopsies it was normal in size, but usually fatty. The iron is in excess, and in striking contrast to cases of secondary anæmia. It is deposited in the outer and middle zones of the lobules, and in two specimens which I examined seemed to have such a distribution that the bile capillaries were distinctly outlined. This is certainly, as Hunter states, a special and characteristic lesion, possibly peculiar to pernicious anæmia. A. J. Scott examined for me the livers in forty-five consecutive autopsies without finding (except in pernicious anæmia) this special distribution of pigment.

The spleen shows no important changes. In one of Palmer Howard's cases the organ weighed only an ounce and five drachms. The iron pigment is usually in excess. The lymph glands may be of a deep red color. The amount of iron pigment is increased in the kidneys, chiefly in the convoluted tubules. The bone marrow, as pointed out by H. C. Wood, may resemble that of a child. This observation has been repeatedly confirmed, but the condition does not appear to be constant. Changes in the ganglion cells of the sympathetic have been reported on several occasions. Lichtheim has found sclerosis in the posterior columns of the cord, which he thinks secondary to the anæmia, and a similar change has been met with in two recent cases by Morris Lewis and Burr.

Symptoms.—The patient may have been in previous good health, but in many cases there is a history of gastro-intestinal disturbance, mental shock, or worry. The description given by Addison presents the chief

features of the disease in a masterly manner. "It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to the earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness in attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless, the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitations are produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles; the debility becomes extreme—the patient can no longer rise from bed; the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking contrast to the failure and exhaustion observable in every other respect."

The Blood.—The corpuscles may sink to one fifth or less of the normal number. They may sink to 500,000 per cubic millimetre, and in a case of Quincke's the number was reduced to 143,000 per cubic millimetre. The hæmoglobin is relatively increased, so that the individual globular richness is plus, a condition exactly the opposite to that which occurs in chlorosis, in which the corpuscular richness in coloring matter is minus. The relative increase in the hæmoglobin is probably associated with the average increase in the size of the red blood-corpuscles. The accompanying chart illustrates these points. Microscopically the red blood-corpuscles present a great variation in size, and there can be seen large giant forms, megalocytes, which are often ovoid in form, measuring eight, eleven, or even fifteen micromillimetres in diameter, a circumstance which Henry regards as indicating a reversion to a lower type. Laache thinks these pathognomonic, and they certainly form a constant feature. There are also small round cells, microcytes, from two to six micromillimetres in diameter, and of a deep red color. The corpuscles show a remarkable irregularity in form, elongated and rodlike or pyriform; one end of a corpuscle may retain its shape while the other is narrow and extended. To this condition of irregularity Quincke gave the name poikilocytosis. The leucocytes are generally diminished in number, and the relative percentage of the mononuclear elements is somewhat higher than in normal blood.

Nucleated red blood-corpuscles are constantly present, as pointed out by Ehrlich. Besides the ordinary form, which is of the same size as the common corpuscle and which has a small, deeply stained nucleus (normo-

blasts), there are very large forms with large, palely staining nuclei (gigantoblasts), which resemble somewhat the larger megalocytes. Ehrlich re-

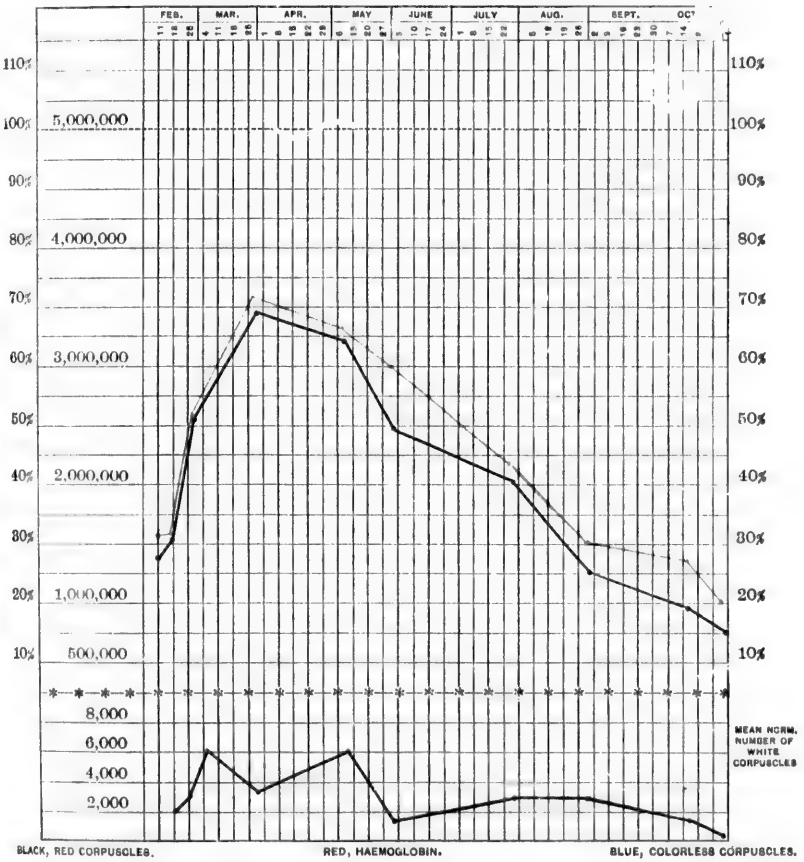


CHART XVIII.—Pernicious anæmia.

gards the presence of these as almost distinctive of progressive pernicious anæmia; they are only found here and in the later stages of leukæmia. The blood-plates are either absent or very scanty.

The cardio-vascular symptoms are important and are noted in the description given above. Hæmic murmurs are constantly present. The larger arteries pulsate visibly and the throbbing in them may be distressing to the patient. The pulse is full and frequently suggests the water-hammer beat of aortic insufficiency. The capillary pulse is frequently to be seen. The superficial veins are often prominent, and in two cases I have seen well-marked pulsation in them. Hæmorrhages may occur, either

in the skin or from the mucous surfaces. Retinal hæmorrhages are common. There are rarely symptoms in the respiratory organs.

Gastro-intestinal symptoms, such as dyspepsia, nausea, and vomiting, may be present throughout the disease. Diarrhœa is not infrequent. The urine is usually of a low specific gravity and sometimes pale, but in other instances it is of a deep sherry color, shown by Hunter and Mott to be due to great excess of urobilin. Fever is a variable symptom. For weeks at a time the temperature may be normal, and then irregular pyrexia may develop. Nervous symptoms may occur, numbness and tingling, and occasionally symptoms resembling those of tabes. Lepine reports a case of extensive paralysis.

Diagnosis.—From chlorosis the disease is readily distinguished. I have not seen a case in which the two diseases could have been confounded. Two points in the blood examination are of importance—namely, the relative increase in the hæmoglobin and the presence of the large forms of nucleated red blood-corpuscles, the gigantoblasts of Ehrlich. Poikilocytosis may occur in any severe anæmia. The separation of the different clinical forms above referred to can usually be made. The profound secondary anæmia of cancer of the stomach may sometimes be puzzling, but the skin is rarely, if ever, lemon-tinted, and the blood has the characteristics of a secondary, not a primary anæmia.

Prognosis.—In the true Addisonian cases the outlook is bad, though of late years on the arsenic treatment the proportion of recovery is increased. My personal experience is as follows: Of the 27 cases 4 are now under observation, 2 of these having recovered with arsenic. Of the remaining 23 the following statement may be made: Four of the 5 post-partum cases recovered, and when I left Montreal 3 of these cases had remained in good health for several years. Of the remaining 18 cases 2 were lost sight of; 1 had improved very much. The remaining 16 are dead. Six of these fatal cases recovered from the first attack; one had an interval of nearly three years, and another nearly two years, before the return. I know of no instance in a male in which the recovery has lasted for five years. In Pye-Smith's article in Guy's Hospital Reports, he mentions twenty cases of recovery. Hale White, in a recent article, states that one of these cases, treated by arsenic in 1880, remained alive and well January, 1891. One of my patients made an apparently complete recovery and resumed active business and political duties. So characteristic are recurrences in this affection that Stephen Mackenzie, in his recent lectures, considered them under a separate heading of relapsing pernicious anæmia.

Treatment of Anæmia.—*Secondary Anæmia.*—The traumatic cases do best, and with plenty of good food and fresh air the blood is readily restored. The extraordinary rapidity with which the normal percentage of red blood-corpuscles is reached without any medication whatever is an important lesson. The cause of the hæmorrhage should be sought and the necessary indications met. The large group depending

on the drain on the albuminous materials of the blood, as in Bright's disease, suppuration, and fever, is difficult to treat successfully, and so long as the cause keeps up it is impossible to restore the normal blood condition. The anæmia of inanition requires plenty of nourishing food. When dependent on organic changes in the gastro-intestinal mucosa not much can be expected from either food or medicine. In the toxic cases due to mercury and lead, the poison must be eliminated and a nutritious diet given with full doses of iron. In a great majority of these cases there is deficient blood formation, and the indications are briefly three—plenty of food, an open-air life, and iron. As a rule it makes but little difference what form of the drug is administered.

The treatment of *chlorosis* affords one of the most brilliant instances—of which we have but three or four—of the specific action of a remedy. Apart from the action of quinine in malarial fever, and of mercury and iodide of potassium in syphilis, there is no other remedy the beneficial effects of which we can trace with the accuracy of a scientific experiment. It is a minor matter *how* the iron cures chlorosis. In a week we give to a case as much iron as is contained in the entire blood, as even in the worst case of chlorosis there is rarely more than a deficit of two grammes of this metal. Iron is present in the fæces of chlorotic patients before they are placed upon any treatment, so that the disease does not result from any deficiency of available iron in the food. Bunge believes that it is the sulphur which interferes with the digestion and assimilation of this natural iron. The sulphides are produced in the process of fermentation and decomposition in the fæces, and interfere with the assimilation of the normal iron contained in the food. By the administration of an inorganic preparation of iron with which these sulphides combine the natural organic combinations in the food are spared. In studying a number of charts of chlorosis it is seen that there is an increase in the red blood-corpuscles under the influence of the iron, and in some instances the globular richness rises above normal. The increase in the hæmoglobin is slower and the maximum percentage may not be reached for a long time. I have for years in the treatment of chlorosis used with the greatest success Blaud's pills, made and given according to the formula in Niemeyer's text-book, in which each pill contains 2 grs. of the sulphate of iron. During the first week one pill is given three times a day. In the second week, two pills; in the third week, three pills, three times a day. This dose should be continued for four or five weeks, at least, before reduction. An important feature in the treatment of chlorosis is to persist in the use of the iron for at least three months, and if necessary subsequently to resume it in smaller doses, as recurrences are so common. The diet should consist of good, easily digested food. Special care should be directed to the bowels, and if constipation is present a saline purge should be given each morning. Such stress does Sir Andrew Clark lay on the importance of constipation in chlorosis that he states that if limited to the choice of one drug in the treatment of the

disease he would choose a purgative. Dilute hydrochloric acid, manganese, phosphorus, and oxygen have been recommended.

Treatment of Pernicious Anæmia.—Since the introduction by Byrom Bramwell of arsenic in this affection a large number of cases have been temporarily, a few permanently, cured by it. It should be given as Fowler's solution in increasing doses. It is usually well borne, and patients, as a rule, take up to twenty minims three times a day without any disturbance. I usually begin with three minims and increase to five at the end of the first week, to ten at the end of the second week, to fifteen at the end of the third week, and, if necessary, increase to twenty or twenty-five. In a case in which the recovery persisted for nearly three years, the dose was gradually increased to thirty minims. These patients seem to bear the arsenic extremely well. It is sometimes better borne as arsenious acid in pill form. Vomiting and diarrhœa are rare; occasionally puffiness of the face is produced, and in some cases pigmentation of the skin.

Rest in bed and a light but nutritious diet (giving the food in small amounts and at fixed intervals) are the first indications. I always prefer to begin the treatment of a case of pernicious anæmia, whatever the grade may be, with rest in bed as one of the essential elements. Massage will also be found very beneficial. I have abandoned the use of rectal injections of dried blood. Iron seems to have no action in this form, but in a case in which the arsenic disagrees it may be tried.

II. LEUKÆMIA.

Definition.—An affection characterized by persistent increase in the white blood-corpuscles, associated with enlargement, either alone or together, of the spleen, lymphatic glands, or bone marrow.

The disease was described almost simultaneously by Virchow and by Bennett, who gave to it the name leucocythæmia.

Etiology.—We know nothing of the conditions under which the disease develops. It is not uncommon on this continent. Of 17 cases of which I have notes, 11 occurred in Montreal, 2 in Philadelphia, and 4 within the past two years at the Johns Hopkins Hospital. It does not seem more frequent in the southern parts of the country.

The disease is most common in the middle period of life. The youngest of my cases was a child of eight months, and cases are on record of the disease as early as the eighth or tenth week. It may occur as late as the seventieth year. Males are more prone to the affection than females. Of my cases 11 were in males and 6 in females. Birch-Hirschfeld states that of 200 cases collected from the literature, 135 were males and 65 females.

A tendency to hæmorrhage has been noted in many cases, and some of the patients have suffered repeatedly from nose-bleeding. In women

the disease is most common at the climacteric. There are instances in which it has developed during pregnancy. The case described by J. Chalmers Cameron, of Montreal, is in this respect remarkable, as the patient passed through three pregnancies, bearing on each occasion non-leukæmic children. The case is interesting, too, as showing the hereditary character of the affection, as the grandmother and mother, as well as a brother, suffered from symptoms strongly suggestive of leukæmia. One of the patient's children had leukæmia before the mother showed any signs, and a second died of the disease. At the last report this patient had gradually recovered from the third confinement and the red blood-corpuscles had risen to 4,000,000 per cubic millimetre, and the ratio of white to red 1 to 200. Sänger has reported a case in which a healthy mother bore a leukæmic child.

Malaria is believed by some to be an etiological factor. Of 150 cases analyzed by Gowers, there was a history of malaria in 30; in my series there was a history in at least 7. Syphilis appears in some cases to have been closely associated with the disease. The disease has followed injury or a blow.

The lower animals are subject to the affection, and cases have been described in horses, dogs, oxen, cats, swine, and mice.

Morbid Anatomy.—The wasting may be extreme, and dropsy is sometimes present. There is in many cases a remarkable condition of polyæmia; the heart and veins are distended with large blood-clots. In Case XI of my series the weight of blood in the heart chambers alone was 620 grammes. There may be remarkable distention of the portal, cerebral, pulmonary, and subcutaneous veins. The blood is usually clotted, and the enormous increase in the leucocytes gives a pus-like appearance to the coagula, so that it has happened more than once, as in Virchow's memorable case, that on opening the right auricle the observer at first thought he had cut into an abscess. The coagula have a peculiar greenish color, somewhat like the fat of a turtle. The alkalinity of the blood is diminished. The fibrin is increased. The character of the corpuscles will be described under the symptoms. Charcot's octohedral crystals separate from the blood after death. The specific gravity of the blood is somewhat lowered. There may be pericardial ecchymoses.

The spleen in the great majority of cases is enlarged. Strong adhesions may unite it to the abdominal wall, the diaphragm, or the stomach. The capsule may be thickened. The vessels at the hilus are enlarged; the weight may range from two to eighteen pounds. The organ is in a condition of chronic hyperplasia. It cuts with resistance, has a uniformly reddish-brown color, and the Malpighian bodies are invisible. Grayish-white, circumscribed, lymphoid tumors may occur throughout the organ, contrasting strongly with the reddish-brown matrix. In the early stage the swollen spleen pulp is softer, and it is stated that rupture has occurred from the intense hyperæmia. Enlargement of the lymphatic glands may

occur, either in conjunction with splenic enlargement or alone. In only one of my cases was the enlargement notable. In the cases of lymphatic leukæmia the cervical, axillary, mesenteric, and inguinal groups may be much enlarged, but the glands are usually soft, isolated, and movable. They may vary considerably in size during the course of the disease. The tonsils and the lymph follicles of the tongue, pharynx, and mouth may be enlarged.

In the majority of cases the bone marrow is involved with the spleen, the lieno-medullary form of the Germans. The marrow may be involved alone, forming a pure myelogenous leukæmia. Instead of a fatty marrow, the medulla of the long bones may resemble the consistent matter which forms the core of an abscess, or it may be dark brown in color. In Ponfick's case there were hæmorrhagic infarctions. There may be much expansion of the shell of bone and localized swellings which are tender and may even yield to firm pressure. Histologically, there are found in the medulla large numbers of nucleated red corpuscles in all stages of development, numerous cells with eosinophilic granules, and also many cells corresponding to the *myelocytes* found in the blood. Large mononuclear cells in the process of division by karyokinesis may be abundant. Polynuclear leucocytes are also present, as well as a certain number of small mononuclear elements.

The thymus is rarely involved, though it has been enlarged in some of the cases of acute lymphatic leukæmia.

In a few instances there have been leukæmic enlargements in the solitary and agminated glands of Peyer. In a case of Willcocks there were growths on the surface of the stomach and gastro-splenic omentum.

The liver may be enlarged, and in a case described by Welch it weighed over thirteen pounds. The enlargement is usually due to a diffuse leukæmic infiltration. The columns of liver cells are widely separated by leucocytes, which are partly within and partly outside the lobular capillaries. There may be definite leukæmic growths.

There are rarely changes of importance in the lungs. The kidneys are often enlarged and pale, the capillaries may be distended with leucocytes, and leukæmic tumors may occur. The skin may be involved, as in a case described by Kaposi.

Leukæmic tumors in the organs are not common. They were present in only one of the twelve autopsies in my series. In 159 cases collected by Gowers there were only thirteen instances of leukæmic nodules in the liver and ten in the kidneys. These new growths probably develop from leucocytes which leave the capillaries. Bizzozero has shown that the cells which compose them are in active fission.

Symptoms.—The onset is insidious, and, as a rule, the patient seeks advice for progressive enlargement of the abdomen and shortness of breath, or for the enlarged glands or the pallor, palpitation, and other symptoms of anæmia. Bleeding at the nose is common. Gastro-intestinal

symptoms may precede the onset. Occasionally the first symptoms are of a very serious nature. In one of the cases of my series the boy played lacrosse two days before the onset of the final hæmatemesis, and in another case, a girl who had, it was supposed, only a slight chlorosis, died of fatal hæmorrhage from the stomach before any suspicion had been aroused as to the true condition.

Blood.—In all forms of the disease the diagnosis must be made by the examination of the blood, as it alone offers distinctive features. In the normal blood Ehrlich recognizes the following varieties of colorless elements: (*a*) Lymphocytes—small cells about the size of a red blood-corpuscle, and probably derived from the lymphatic glands, which have a single large, round, deeply staining nucleus, surrounded by a narrow rim of non-granular protoplasm. (*b*) Large mononuclear leucocytes—cells several times as large as the red blood-corpuscle, with an oval or elliptical nucleus and a relatively larger amount of ungranulated protoplasm. (*c*) Transitional forms—cells which resemble the last variety, but have indentations and irregularities in the nucleus. (*d*) Polynuclear leucocytes—these are about the same size or a little smaller than the last variety. The nucleus is a long, deeply staining body which is bent and twisted on itself into irregular shapes. The protoplasm of these cells is filled with granules, which are stained, not by acid or basic coloring matters alone, but only by a combined fluid. The granules are therefore termed neutrophilic, and the name "neutrophiles" is given to these cells. (*e*) Cells about the same size as the last, but containing large, highly refractile, fat-like granules, which have an affinity for acid coloring matters. On account of their affinity for eosin, Ehrlich terms them *eosinophiles*. In normal blood these cells occur in a definite proportion to each other; the lymphocytes fifteen to thirty per cent, the polynuclear sixty-five to eighty per cent, the mononuclear and transitional forms about six per cent, and the eosinophiles two to four per cent.

The most striking change in the more common form, the lienomyelogenic, is the increase in the colorless corpuscles. The average number of white per cubic millimetre is estimated at about 6,000; thus the proportion of white to red is 1 to 500—1,000. In leukæmia the proportion may be 1 to 10, or 1 to 5, or the ratio may reach 1 to 1. There are instances on record in which the number of leucocytes has exceeded that of the red corpuscles.

The character of the cells in splenic myelogenous leukæmia is as follows: The lymphocytes are little, if at all, increased; relatively they are greatly diminished. The eosinophiles are present in normal or increased relative proportion, so that there is a great total increase, and their presence is a striking feature in the stained blood-slide. The polynuclear neutrophiles may be in normal proportion; more frequently they are relatively diminished, and in the latter stages they may form but a small proportion of the colorless elements. The most characteristic feature

of the blood in this form of leukæmia is the presence of cells which do not occur in normal blood. They appear to be derived from the marrow, and are called by Ehrlich *myelocytes*. They are as large or even larger than the large mononuclear leucocytes, and are similar to them in appearance, but differ from them in the fact that the protoplasm is filled with the fine neutrophilic granules. Müller has recently found many large mononuclear elements with karyokinetic figures in leukæmic blood and in the marrow.

Nucleated red blood-corpuscles are present, usually in considerable numbers. There is, as a rule, only a moderate reduction in the number of red blood-corpuscles, rarely under two million per cubic millimetre. The hæmoglobin is usually reduced in a somewhat greater proportion. The accompanying blood chart is from a case of leukæmia with an enormously enlarged spleen.

The histological characters of the blood in acute lymphatic leukæmia differ materially. The increase in the colorless elements is never so great as in the preceding form; a proportion of one to ten would be extreme. This increase takes place solely in the lymphocytes, all other forms of leucocytes being present in greatly diminished relative proportion. In Uthemann's case ninety-three per cent of all the leucocytes were lymphocytes. Eosinophiles and nucleated red corpuscles are rare. Myelocytes are not present. As occasionally combined forms of leukæmia may occur, so undoubtedly variations from these two types of blood may be met with, and in a case of acute leukæmia observed at the Johns Hopkins Hospital, in which glands, marrow, and spleen were affected, there was present, besides a large proportion of lymphocytes and myelocytes, a considerable number of large mononuclear leucocytes. Among other points about leukæmic blood may be mentioned the feebleness of the amœboid movement, as noted by Cafafy, which may be accounted for by the large number of mononuclear elements present, the polynuclear alone possessing this power. The blood-plates exist in variable numbers; they may be remarkably abundant. The fibrin network between the corpuscles is usually thick and dense. In blood-slides which are kept for a short time, Charcot's octohedral crystals separate, and in the blood of leukæmia the hæmoglobin shows a remarkable tendency to crystallize.

The pulse is usually rapid, soft, compressible, but often full in volume. There are rarely any cardiac symptoms. The apex beat may be lifted an interspace by the enlarged spleen. Toward the close, as a consequence of the feeble circulation, œdema may occur in the feet or there may be general anasarca. Hæmorrhage is a common symptom and may be either late or early. Epistaxis is the most frequent form. Hæmoptysis and hæmaturia are rare. Bleeding from the gums may be present. Hæmatemesis proved fatal in two of my cases, and in a third a large cerebral hæmorrhage rapidly killed. The leukæmic retinitis is a part of the hæmorrhagic manifestations.

There are very few pulmonary symptoms. The shortness of breath is due, as a rule, to the anæmia. Toward the end there may be œdema of

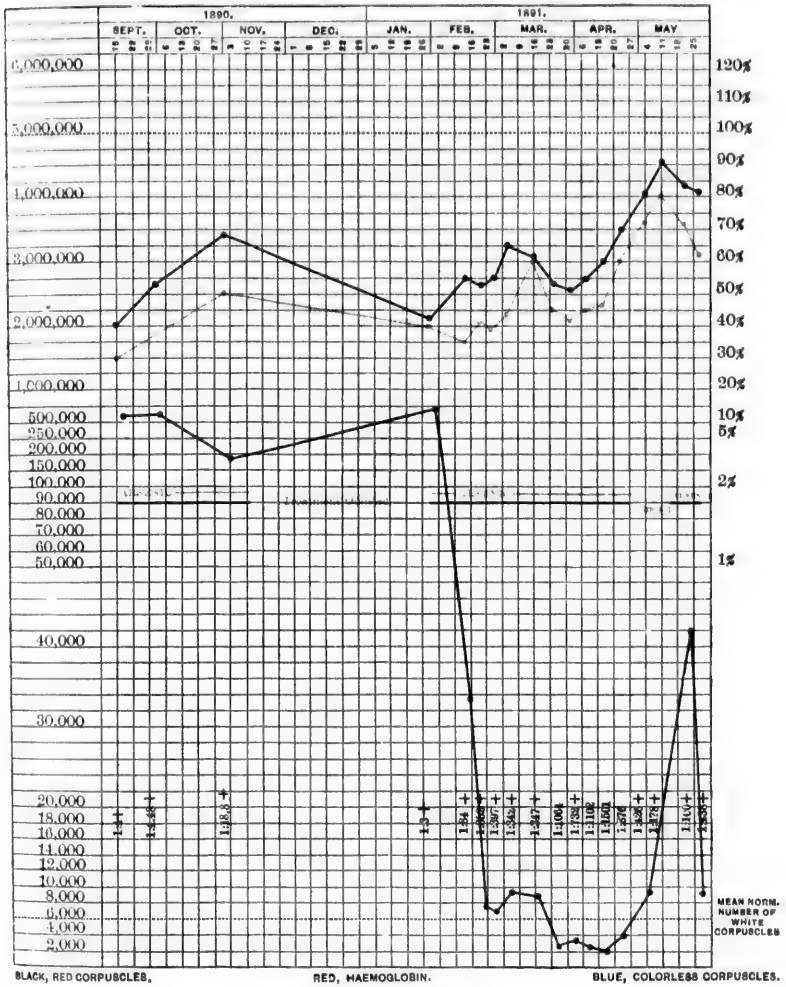


CHART XIX.—Leukæmia.

the lungs or pneumonia may carry off the patient. The gastro-intestinal symptoms are rarely absent. Nausea and vomiting are early features in some cases. Diarrhœa may be very troublesome, even fatal. Intestinal hæmorrhage is not common. There may be a dysenteric process in the colon. Jaundice rarely occurs, though in one case of my series there were recurrent attacks. Ascites may be a prominent symptom, probably due

to the presence of the splenic tumor. A leukæmic peritonitis also may be present, due to new growths in the membranes.

The nervous system is not often involved. Headache, dizziness, and fainting spells are due to anæmia. The patients are usually tranquil and resigned. Sudden coma may follow cerebral hæmorrhage.

The special senses are often affected. There is a peculiar retinitis, due chiefly to the extravasation of blood, but there may be aggregations of leucocytes, forming small leukæmic growths. Optic neuritis is rare. Deafness has frequently been observed; it may appear early and possibly is due to hæmorrhage.

The urine presents no constant changes. The uric acid excreted is always in excess, and possibly, as Salkowski suggests, stands in direct relation to the splenic tumor.

Priapism is a curious symptom which has been present in a large number of cases. It may, as in one of Edes' cases, be the first symptom. Peabody reports a case in which it persisted for six weeks. The cause is not known.

Slight fever is present in a majority of cases. Periods of pyrexia may alternate with prolonged intervals of freedom. The temperature may range from 102° to 103°.

The Spleen.—Gradual increase in volume of this organ is the most prominent symptom in a majority of the cases. Pain and tenderness are common, though the progressive enlargement may be painless. A creaking fremitus may be felt on palpation. The enlarged organ extends downward to the right, and may be felt just at the costal edge, or when large it may extend as far over as the navel. In many cases it occupies fully one half of the abdomen, reaching to the pubes below and extending beyond the middle line. As a rule, the edge, in some the notch or notches, can be felt distinctly. Its size varies greatly from time to time. It may be perceptibly larger after meals. A hæmorrhage or free diarrhœa may reduce the size. The pressure of the enlarged organ may cause distress after eating; in one case it caused fatal obstruction of the bowels. A murmur may sometimes be heard over the spleen, and Gerhardt has described a pulsation in it.

The Lymph Glands.—Lymphatic leukæmia is rare. As mentioned, in but 1 of my series of 17 cases were the glands enlarged; indeed, no instance of pure lymphatic leukæmia has come under my observation. The superficial groups are usually most involved, and even when affected it is rare to see such large bunches as in Hodgkin's disease. External lymph tumors are rare.

The pure myelogenous cases without associated enlargement of the spleen are rare. The most extreme hyperplasia of the bone marrow may exist without any tenderness. Occasionally the sternum, ribs, and flat bones show great irregularity and deformity, owing to definite tumor-like expansions.

Diagnosis.—The recognition of leukæmia can be determined only

by microscopical examination of the blood. The clinical features may be identical with those of ordinary splenic anæmia, or with Hodgkin's disease. An interesting question arises whether real increase in the leucocytes is the only criterion of the existence of the disease. Thus, for instance, in the case whose chart is given, on page 701, the patient came under observation in September, 1890, with 2,000,000 red blood-corpuscles per cubic millimetre, thirty per cent of hæmoglobin, and 500,000 white blood-corpuscles per cubic millimetre—a proportion of one to four. As shown by the chart, throughout September, October, November, and December, this ratio was maintained. Early in January, under treatment with arsenic, the white corpuscles began to decrease and gradually, as shown in the chart, the normal ratio was reached. At this time could it be said that the case was one of leukæmia without increase in the number of leucocytes? The blood examination by Ehrlich's method, as made by Thayer, showed that the characteristic myelocytes, elements which are not present in normal blood, were still present in numbers sufficient, at any rate, to suggest, if the patient had come under observation for the first time, that leukæmia might occur. By Ehrlich's method of blood examination a condition of leucocytosis can readily be distinguished from that of leukæmia, for in all ordinary leucocytoses the increase takes place solely in the polynuclear neutrophilic leucocytes, forming quite a different picture from the characteristic conditions described above.

Prognosis.—Recovery occasionally occurs. A great majority of the cases prove fatal within two or three years. Unfavorable signs are a tendency to hæmorrhage, persistent diarrhœa, early dropsy, and high fever. Remarkable variations are displayed in the course, and a transient improvement may take place for weeks or even months. The pure lymphatic form seems to be of particular malignancy, some cases proving fatal in from six to eight weeks.

Treatment.—Fresh air, good diet, and abstention from mental worry and care, are the important general indications. The *indicatio morbi* cannot be met. There are certain remedies which have an influence upon the disease. Of these, arsenic, given in large doses, is the best. I have repeatedly seen improvement under its use. On the other hand, there are curious remissions in the disease which render therapeutical deductions very fallacious. I have seen such marked improvement without special treatment that the patient, from a bed-ridden, wretched condition, recovered strength enough to enable him to attend to light duties.

Quinine may be given in cases with a malarial history. Iron may be of value in some cases, as may also inhalations of oxygen.

Excision of the leukæmic spleen has been performed twenty-four times, with one recovery—the case of Franzolini. Fussell gives the statistics of 105 cases of splenectomy with 48 deaths. Of the cases of simple hypertrophy, 28 in number, 9 recovered. Of 16 cases of floating spleen, 15 recovered.

III. HODGKIN'S DISEASE.

Definition.—An affection characterized by progressive hyperplasia of the lymph glands, with anæmia, and occasionally the development of secondary lymphoid growths in liver, spleen, and other organs. The disease has also the names *pseudo-leukæmia*, *general lymphadenoma*, and *adénie*.

Hodgkin, the well-known morbid anatomist of Guy's Hospital, first described cases in detail, and by the labors of Wilks, Virchow, Billroth, and Cohnheim the disease attained definite recognition.

Etiology.—A majority of the cases are in young persons. In Gowers' table of 100 cases, 30 were under twenty years, 34 between twenty and forty, and 36 above forty. Three fourths of the cases are in males. In a few instances heredity has been adduced as a possible cause, and antecedent disease, such as syphilis, but this is doubtful. More important is local irritation, upon which Trousseau lays special stress, and gives instances in which chronic irritation of the skin, chronic nasal catarrh, or the irritation of a decayed tooth gave rise to local gland swellings, which preceded a general development of the disease. In a large majority of the cases the disease comes on insidiously, without any recognizable cause.

Morbid Anatomy.—*The Lymph Glands.*—In a few cases the enlarged glands are hard and firm, but in a majority the growth is soft and elastic. In the early stage the individual glands are isolated, not larger than almonds or walnuts, and readily separated and movable. When advanced the glands fuse together, and a group, as in the neck, may form a large tumor, the size of an orange or even of a cocoa-nut. About such masses the capsular tissues are hard and dense, forming a firm investment. A growth may perforate the capsule and invade contiguous parts, such as the muscles, skin, or the solid organs. On section, the tumor has a grayish-white appearance; it is smooth, and of variable consistence, either firm and dry or soft and juicy. Suppuration is most frequently seen when the growth reaches the skin. In the deep glands the formation of pus is rare. Caseation is not common; occasionally there are areas of necrosis very like it. The superficial glands are most often attacked, particularly the cervical groups, and the glands may be traced as continuous chains along the trachea and the carotids, uniting the axillary and mediastinal glands.

The axillary group is involved next in order of frequency, and the masses may pass beneath the pectorals and beneath the scapulæ. The inguinal glands occasionally form very large masses. Of the internal groups, those of the thorax are most often affected, either the chain in the posterior mediastinum or the bronchial group, or those of the anterior mediastinum. The trachea and the aorta with its branches may be completely surrounded by the growths, and be but little compressed. From the anterior mediastinum the masses may perforate the sternum and appear as an external tumor.

Of the abdominal groups, the retroperitoneal is most frequently involved and may form a continuous chain from the diaphragm to the inguinal canals, and extend into the pelvis. The glands may compress the ureters, involve the sacral or lumbar nerves, or compress the iliac veins. Occasionally they adhere to the uterus and broad ligament so as to simulate fibroids. I saw, some years ago, one of the most distinguished gynæcologists of Germany perform laparotomy in a case of this kind, in which the diagnosis of myomatous tumors of the uterus had been made. Occasionally the mesenteric or hepatic lymph glands may form large abdominal tumors.

Histologically the chief change is an increase in the cells, with or without thickening of the reticulum. In the early stage there is simple hyperplasia and the relations of the lymph paths are maintained, but when the glands are greatly enlarged the normal arrangement is disturbed. The reticulum varies extremely; in the softer growths it is expanded and can scarcely be found; in the harder structures the network of fibres is very distinct, and there is probably an increased development of the adenoid tissue.

Spleen.—In seventy-five per cent of the cases collected by Gowers this organ was hypertrophied, and in fifty-six of these cases it presented lymphoid growths. The enlargement is rarely great, and does not approximate to the large leukæmic spleen. The lymphoid tumors form grayish-white bodies ranging in size from a pea to a walnut, and may resemble lymph glands in appearance and consistence. Histologically, they consist of lymph corpuscles in a fibrous reticulum.

The marrow of the long bones may be converted into a rich lymphoid tissue; in a few instances the pyoid form, such as is more common in leukæmia, has been found. The tonsils may be involved and the follicles at the root of the tongue. Occasionally secondary growths are seen in the intestines.

The liver is often enlarged and may present scattered lymphoid tumors. The kidneys are occasionally involved and are the seat of growths similar to those of the spleen and liver. The lungs are occasionally directly attacked from the bronchial glands at the root, and secondary nodules may be found throughout their substance. Pleural effusions are not uncommon. Involvement of the nervous system is rare, but paraplegia may be induced by invasion of the spinal canal. The skin may be the seat of adenoid growths, as in a case reported by Greenfield.

Symptoms.—Enlargement of the glands of the neck, axilla, or groins is usually the first symptom noticed. In a few cases the anæmia and constitutional symptoms attract attention before the glandular involvement is evident. When the trouble begins in the deeper groups, pressure effects may be first noticed; thus, paroxysmal dyspnœa with pain in the chest may result from enlargement of the bronchial glands before any physical signs can be detected. Œdema of the feet and shooting

pains in the nerves were the first symptoms in one case which I dissected for Ross, and in another case at the Montreal General Hospital there was paraplegia from pressure on the cord. Such instances, however, are exceptional, and in the majority of cases the swelling of the superficial glands is the earliest symptom. Epistaxis has occasionally been noted, but not so frequently as in leukæmia. With progressive enlargement of the glands the patient becomes anæmic.

Usually, the cervical group is first affected, and it may be impossible to decide whether the enlargement is syphilitic, tuberculous, or lymphadenomatous. One side is first affected as a rule, and it may be months, or even, as in one of my cases, three years before the affection extends to other groups Ultimately huge tumors may develop, which obliterate the neck and extend upon the shoulders and over the clavicles and sternum. The trachea is surrounded, great dyspnœa is produced, and not infrequently tracheotomy is necessary. In the later stages, the skin becomes involved and ulcerates. The axillary group may form large tumors, which compress the brachial or axillary veins and cause swelling of the arms. The inguinal glands are not so often involved, but may form large or even pendulous tumors

In the thoracic glands, as mentioned, the various groups may be involved and produce pressure upon the veins or upon the trachea. In a case at present under observation the superior cava is completely obliterated and a very extensive collateral circulation has been established by means of the mammary and epigastric veins. The skin over the sternum is a mass of fluctuating veins, some of which contain phleboliths. In the abdomen the mesenteric glands may be enlarged, or more commonly the retroperitoneal group. When the patient is thin there may be no difficulty in detecting these, but in stout persons the diagnosis may be impossible. In connection with the affections of the abdominal glands there may be bronzing of the skin, which was well marked in Case IV of my series. A remarkable feature is the variations in the rate of growth and in the size of the glands. They may reduce rapidly and almost disappear from a region, and before death the tumors may diminish very much. The spleen may be enlarged and readily palpable. The thyroid also may be involved, and in a few instances the thymus has been affected. Though present in a majority of the cases, there may be enormous enlargement of the lymph glands without marked anæmia. In one of my cases the blood-corpuscles did not sink below 4,000,000 per cubic millimetre, and in only one instance have I counted the blood below 2,000,000. The red blood-corpuscles rarely show extreme poikilocytosis. The white corpuscles may be moderately increased and the lymphocytes most abundant. Occasionally the leucocytes are greatly increased and the characters of the blood become those of a lymphatic leukæmia. Nucleated red blood-corpuscles may be present, but not in such numbers as in leukæmia.

Of cardiac symptoms, palpitation is common. Hæmic murmurs are

often heard over the heart. Shortness of breath may be due to the anæmia, to pressure upon the trachea, or, in some instances, to pleuritic effusion associated with mediastinal growths. Fever is observed in nearly all cases; even in the early stages there is slight elevation. It may be of an irregular hectic type, or continuous, with evening exacerbation. Very remarkable are the cases with ague-like paroxysms, which may persist for weeks or months. They were present in Case I of my series. Pel, of Amsterdam, has given a thorough description of these attacks, and Ebstein has described a case under the remarkable title of "Chronic Recurrent Fever, a New Infectious Disease." In his case during nine months the attacks were present for periods of from twelve to fourteen days and alternated with apyrexia for ten or eleven days.

The digestive symptoms are usually not marked. It is not uncommon to find albumen in the urine. Headache, giddiness, and noises in the ear may be associated with the anæmia. Delirium and coma may be present. Deafness may be produced by growth of the adenoid tissue in the pharynx close to the Eustachian tubes. Inequality of the pupils may be present, owing to pressure of the glands on the cervical sympathetic. The skin may show definite secondary lymphatic tumors, bronzing may occur, and occasionally a most intense and troublesome prurigo.

Diagnosis.—A tuberculous adenitis may at first be very difficult to differentiate. The chief points of distinction are as follows: Tuberculous adenitis is more common in the young and involves the submaxillary group of glands more frequently than those of the anterior and posterior cervical triangles, which are usually affected first in Hodgkin's disease. The enlargement may last for years in a group without extending. The bunches are often, when small, welded together and, most important of all, tend to suppurate—a feature rarely seen in true lymphadenoma, except when it has attained very large size. Strict limitation to one side of the neck or to the axilla is suggestive of tuberculous disease rather than lymphadenoma.

There is an acute tuberculous adenitis, which may involve the lymph glands of the neck, producing enormous enlargement. A man, aged twenty-four, was admitted to the General Hospital, Montreal, with great swelling of the cervical glands on both sides, tonsillitis, and sloughing pharyngitis, with irregular fever and diarrhœa. The case was at first regarded as one of Hodgkin's disease. The occurrence of rigors and intermittent pyrexia is in favor of lymphadenoma. There are cases in which it may for a time be impossible to make a diagnosis. When the glands are only moderately enlarged on one side of the neck or axilla, they should be removed, and the diagnosis can then be thoroughly established.

Prognosis.—Recovery is very rare. The course of the disease is extremely variable. Early and rapid growth in the mediastinal groups may produce pressure effects and cause death before the development is extreme. In some cases the enlargements spread rapidly and group after

group becomes involved in a few months. These acute cases may run a course in three or four months. Chronic cases may last for three or four years. Periods of quiescence are not uncommon. The tumors may not only cease to grow, but gradually diminish and even disappear, without special treatment. Usually a cachexia develops, the anæmia progresses, and there are dropsical symptoms. The mode of death is usually by asthenia; less commonly by pressure from a tumor; and occasionally by coma.

Treatment.—When small and localized the glands should be removed. Local applications are of doubtful benefit. I have never seen special improvement follow the persistent use of iodine or the various ointments.

Arsenic has a positive value in the disease. It should be given in increasing doses, and stopped when unpleasant effects are manifested. The results have in many instances been striking. Due allowance must be made for the fluctuations in the size of the growths which occur spontaneously. I have seen no ill effects from the administration of Fowler's solution for months at a time, and many patients have taken from fifteen to twenty minims three times a day for weeks, and in some instances for months. Recoveries have been reported under this treatment. Personally, no instance of recovery has come under my notice in the cases of which I have notes. Phosphorus is recommended by Gowers and Broadbent, and should be used if the arsenic is not well borne. Quinine, iron, and cod-liver oil are useful as tonics. Every possible means must be taken to support the patient's strength.

IV. ADDISON'S DISEASE.

Definition.—A constitutional affection characterized by asthenia, depressed circulation, irritability of the stomach, and pigmentation of the skin. In a majority of the cases it is associated with tuberculous disease of the adrenals, in other instances with wasting of these organs or with changes in the abdominal sympathetic system.

The recognition of the disease is due to Addison, of Guy's Hospital, whose monograph on The Constitutional and Local Effects of Disease of the Suprarenal Capsules was published in 1855.

Etiology.—Males are more frequently attacked than females. In Greenhow's analysis of 183 cases 119 were males and 64 females. A majority of the cases occur between the twentieth and the fortieth year. A congenital case has been described in which the skin had a yellow-gray tint. The child lived for eight weeks, and post mortem the adrenals were found to be large and cystic. Injury, such as a blow upon the abdomen or back, and caries of the spine have in many cases preceded the attack. The disease is rare in America. Eight cases have come under my personal observation, either clinically or anatomically.

Morbid Anatomy and Pathology.—There is rarely emaciation or anæmia. In a great majority of the cases the adrenals are affected. There may be (*a*) atrophy of one or both glands, due to an interstitial cirrhosis, of which cases have been described by Hadden and Goodhart. (*b*) Tuberculosis, which is the common condition. The capsules are thickened and present firm caseous masses, surrounded by connective tissue. There is usually much fibrous thickening and matting of the adjacent structures, and the affection has definitely been shown to be tuberculous. Tuberculous lesions are common in other parts, particularly in the lungs, though in a number of the cases tuberculosis has been limited to the adrenals. (*c*) There may be malignant disease of the adrenals, which has been present in a few instances of genuine Addison's disease. Among other anatomical features the condition of the abdominal sympathetic has been specially studied. The nerve-cells of the semilunar ganglia have been described as degenerated and deeply pigmented, and the nerves sclerotic. The ganglia are not uncommonly entangled in the cicatricial tissue about the adrenals. The spleen has occasionally been found enlarged; the thymus may persist and be larger than normal.

It is difficult to explain satisfactorily all the symptoms of this remarkable disease. The theories which have been advanced are briefly as follows: (*a*) That the disease depended upon the loss of function of the adrenals. This was the view of Addison. It is held that the blood is gradually poisoned by the retention of some material, the destruction or alteration of which is a function of the suprarenals; (*b*) that it is an affection of the abdominal sympathetic system, induced most commonly by disease of the adrenals, but also by other chronic affections which involve the solar plexus and its ganglia. According to this view, it is an affection of the nervous system, and the pigmentation has its origin in changes induced through the trophic nerves. The pronounced debility is the outcome of disturbed tissue metabolism, and the circulatory, respiratory, and digestive symptoms are due to implication of the pneumogastric. The changes found in the abdominal sympathetic are held to support this view, and its advocates urge the occurrence of pigmentation of the skin in tuberculosis of the peritonæum, cancer of the pancreas, or aneurism of the abdominal aorta. Opposed to it are the facts that the lesions described in the sympathetic system are indefinite, and identical changes occur without the symptoms of Addison's disease.

Symptoms.—In the words of Addison the characteristic symptoms are "anæmia, general languor or debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of color in the skin."

The pigmentation is the symptom which, as a rule, first attracts attention. The grades of coloration range from a light yellow to a deep brown, or even black. In typical cases it is diffuse, but always deeper on the exposed parts and in the regions where the normal pigmentation is

most intense. At first it may be confined to the face and hands. Occasionally it is absent. Patches of atrophy of pigment, leucoderma, may occur. The pigmentation is found on the mucous membranes of the mouth, conjunctivæ, and vagina. A patchy pigmentation of the serous membranes has often been found. The anæmia, upon which Addison laid stress, is of a moderate grade. It was not present in a marked degree in any of my cases.

Gastric disturbances are common; nausea and vomiting may be early and prominent symptoms; diarrhœa, too, is frequent, and may come on without cause. The pulse is small and rapid, and the heart's action feeble. Sometimes there is a special liability to syncope. One of the most pronounced features of the disease is the profound asthenia, which is out of all proportion to the general condition. The patient complains of a lack of energy, both mental and bodily; the least exertion is an effort, and may be followed by giddiness or noises in the ears. Headache is a frequent symptom. With the advancement of the disease the prostration becomes more marked, the patient remains in bed, the voice gets weak, the intelligence dulled, and death occurs either by syncope or gradual asthenia. Occasionally there are convulsions. The urine is usually normal. Polyuria has been described. The urinary pigments have been found increased.

Diagnosis.—Pigmentation of the skin is not confined to Addison's disease. The following are the conditions which may give rise to an increase in the pigment:

(1) Abdominal growths—tubercle, cancer, or lymphoma. In tuberculosis of the peritonæum pigmentation is not uncommon.

(2) Pregnancy, in which the discoloration is usually limited to the face, the so-called *masque des femmes enceinte.* Uterine disease is a common cause of a patchy melasma.

(3) Hepatic disease, which may induce definite pigmentation, as in the diabetic cirrhosis. More commonly in overworked persons of constipated habit and with sluggish livers there is a patchy staining about the face and forehead.

(4) The vagabond's discoloration, caused by the irritation of lice and dirt, which may reach a very high grade, and has sometimes been mistaken for Addison's disease.

(5) In rare instances there is deep discoloration of the skin in melanotic cancer, so deep and general that it has been confounded with *melasma suprarenale.*

(6) In certain cases of exophthalmic goitre abnormal pigmentation occurs, as noted by Drummond and others.

In any case of unusual pigmentation these various conditions must be sought for, and the diagnosis of Addison's disease is scarcely justifiable without the asthenia. In many instances it is difficult early in the disease to arrive at a definite conclusion. The occurrence of

fainting fits, of nausea, and gastric irritability is an important indication.

Prognosis.—The disease is usually fatal. The cases in which the bronzing is slight or does not occur run a more rapid course. There are occasionally acute cases which, with great weakness, vomiting, and diarrhœa, prove fatal in a few weeks. In a few cases the disease is much prolonged, even to six or ten years. In rare instances recovery has taken place, and periods of improvement, lasting many months, may occur.

Treatment.—The causal indications cannot be met. When there is profound asthenia the patient should be confined to bed, as fatal syncope may at any time occur. In three of my cases death was sudden. When anæmia is present iron may be given in full doses. Arsenic and strychnia are useful tonics. For the diarrhœa large doses of bismuth should be given; for the irritability of the stomach, creosote, hydrocyanic acid, ice, and champagne. The diet should be light and nutritious. Many patients thrive best on a strictly milk diet.

V. DISEASES OF THE THYROID GLAND.

Goitre.

Definition.—Hypertrophy of the thyroid gland, occurring sporadically or endemically.

In this country sporadic cases are common. Endemically it is found particularly in the mountainous regions of Switzerland and in parts of Italy. No satisfactory explanation has been given of the existence of the disease in this form.

Anatomically the following varieties may be distinguished: (*a*) Parenchymatous, in which the enlargement is general and the follicles, usually newly formed, contain a gelatinous colloid material. (*b*) Vascular, in which the enlargement is chiefly due to dilatation of the blood-vessels without the new formation of glandular tissue. (*c*) Cystic goitre, in which the enlarged gland is occupied by large cysts, the walls of which often undergo calcification.

Symptoms.—The enlargement may be uniform throughout the entire gland, or affect only one lobe, or the isthmus alone. When small, a goitre causes no inconvenience. In its growth it may compress the trachea, causing dyspnœa, or may pass beneath the sternum and compress the veins. These, however, are exceptional circumstances, and in a large proportion of all cases no serious symptoms are noted. The affection usually comes under the care of the surgeon. Sudden death occasionally occurs in large bronchoceles. In some instances it may be difficult to determine the cause and it has been thought to be associated with pressure on the vagi. I have reported an instance in which it resulted from hæmor-

rhage into the gland and into the adjacent tissues. The blood passed into the cellular tissues of the neck and into the sternum, covering the aorta and pericardium.

Tumors of the Thyroid.

These are very varied. (*a*) Adenomata, either simple or malignant. The latter may form extensive metastases. A case is reported by Hayward in which growths resembling thyroid tissue occurred in the lungs and various bones of the body. (*b*) Cancer, of which several forms have been described. (*c*) Sarcoma. All of these have a surgical rather than a medical interest.

It may be mentioned that the aberrant or accessory thyroid gland may form large tumors in the mediastinum or in the pleura. I have reported two cases of this kind,* and an instance is on record in which an enormous cystic accessory thyroid occupied the entire right pleura.

Exophthalmic Goitre (*Graves's Disease; Basedow's Disease*).

Definition.—A disease of unknown origin, characterized by exophthalmos, enlargement of the thyroid, and functional disturbance of the vascular system.

Etiology.—The disease is rare in men. The age of onset is usually from the twentieth to the thirtieth year. It is sometimes seen in several members of the same family. Worry, fright, and depressing emotions precede the development of the disease in a number of cases.

Morbid Anatomy and Pathology.—No constant changes have been found in exophthalmic goitre. Special attention has been paid to the condition of the sympathetic system, as the rapid action of the heart and dilatation of the vessels has been attributed to paralysis of the sympathetic fibres, particularly the vaso-dilators. This view has found many supporters, but neither in the ganglia nor in the nerves are there any changes which can be regarded as constant and peculiar (Hale White). On the other hand, many features of the disease are explicable on the view that it is an affection of the medulla oblongata, and Hale White has reported a case dying of an acute intercurrent disease in which there were hæmorrhages in the floor of the fourth ventricle. The vascular and nervous features might be due to a lesion of this part; but it is difficult on any theory to explain all the symptoms of the disease and to bring into line the mental and vascular phenomena, the exophthalmos and the goitre.

Symptoms.—Acute and chronic forms may be recognized. In the acute form the disease may develop with great rapidity. In a patient of J. H. Lloyd's, of Philadelphia, a woman, aged thirty-nine, who had been considered perfectly healthy, but whose friends had noticed that for

* Medical News, 1890.

some time her eyes looked rather prominent, was suddenly seized with intense vomiting and diarrhœa, rapid action of the heart, and great throbbing of the arteries. The eyes were prominent and staring and the thyroid gland was found much enlarged and soft. The gastro-intestinal symptoms continued, the pulse became more rapid, the vomiting was incessant, and the patient died on the third day of the illness; only the abdominal and thoracic organs could be examined and no changes were found. Two rapidly fatal cases occurred at the Philadelphia Hospital, one of which, under F. P. Henry's care, had marked cerebral symptoms. More frequently the onset is gradual and the disease is chronic. The three characteristic symptoms vary a good deal in their onset. Cardiac and vascular symptoms are usually first to develop and the patient complains of palpitation with breathlessness, and on examination the impulse is found to be increased in force, the apex beat is in normal position, the carotids throb, and the abdominal aorta pulsates visibly. This is one of the conditions in which the capillary pulse and the pulsation in the veins of the hands are occasionally seen. The pulse-rate at first may not be more than 95 or 100, but when the disease is established may reach 140 or 160. Any emotional excitement sets the heart beating with great intensity, and on exposure of the skin of the upper part of the chest a transient hyperæmia is seen. Soft murmurs are not uncommon at the base of the heart. In the long-standing cases the heart may be hypertrophied and the sounds very intense. In rare instances they may be heard some distance from the patient; according to Graves, as far as four feet.

Exophthalmos usually follows the vascular disturbance. It is readily recognized by the protrusion of the balls, and partly by the fact that the lids do not completely cover the sclerotics, so that a rim of white is seen above and below the cornea. The protrusion may become very great and the eye may even be dislocated from the socket. The vision is normal. Graefe noted that when the eyeball is moved downward the upper lid does not follow it as in health. This is known as Graefe's sign. The palpebral aperture is wider than in health, owing to spasm or retraction of the upper lid (Stellwag's sign). Changes in the pupils and in the optic nerves are rare. Pulsation of the retinal arteries is common.

The enlargement of the thyroid commonly develops with the exophthalmos. It may be general or in only one lobe, and is rarely as large as in ordinary goitre. The vessels are usually much dilated, and the whole gland may be seen to pulsate. A thrill may be felt on palpation and on auscultation a loud systolic murmur, or more commonly a *bruit de diable*. Among other symptoms which may develop are anæmia, emaciation, and slight fever. Attacks of vomiting and diarrhœa may occur. Tremor is present in many cases, involving the hands, and is usually very fine. The greatest complaint is of the unpleasant throbbing in the arteries, often accompanied with unpleasant flushes of heat and profuse perspirations. Skin symptoms are not infrequent—pigmentation, patches of leucoderma,

or atrophy of pigment, and urticaria. In the very acute case above referred to, urticaria was a prominent symptom. Irritability of temper, change in disposition, and great mental depresssion have been described. An important complication is acute mania, in which the patient may die in a few days. Symptoms of general paresis have been noted in a few cases. A feature of interest noted by Charcot is the great diminution in the electrical resistance, which may be due to the saturation of the skin with moisture owing to the vaso-motor dilatation (Hirt). Bryson has noted the fact that the chest expansion may be greatly diminished.

The course of the disease is usually chronic, lasting several years. After persisting for six months or a year the symptoms may disappear. There are remarkable instances in which the symptoms have come on with great intensity, following fright, and have disappeared again in a few days. A certain proportion of the cases recover, but when the disease is well developed recovery is rare.

Treatment.—Medicinal measures are notoriously uncertain. The combination of digitalis and iron may be tried, and, when there is anæmia, often does good. I have never seen any advantage from the use of aconite or veratrum viride. The tincture of strophanthus will sometimes reduce the rapidity of the heart's action. Ergot is warmly recommended by some writers. Belladona gives relief occasionally, and should be administered until the dryness of the throat is obtained. No measures are so successful as rest in bed with an ice-bag or Leiter's tube applied occasionally over the heart, or, what is sometimes more agreeable, over the lower part of the neck and manubrium sterni. I have known the pulse to be reduced in this way from 140 to 90. Electricity has been much lauded and instances of cure have been reported. In many cases temporary improvement certainly follows the use of the galvanic current, the cathode being placed at the back of the neck and the anode along the course of the sympathetic or over the heart. Treatment of the thyroid gland itself is rarely successful, and the operative measures have not been very satisfactory. Ligation of the arteries of the thyroid has been tried.

Myxœdema.

Definition.—A constitutional affection, characterized clinically by a myxœdematous condition of the subcutaneous tissues and mental failure, and anatomically by atrophy of the thyroid gland. The disease was described by Sir William Gull as a cretinoid change, and by Ord as a special disease, to which he gave the above name.

Clinical Forms.—Three groups of cases may be recognized: (*a*) *Congenital form*, or sporadic cretinism. In these cases there is congenital absence of the thyroid, and the child is a dwarf, having a thick neck, short arms and legs, and prominent abdomen. The face is large, the lips are

thick, the tongue is large and usually protrudes. The mental condition is that of imbecility or idiocy.

(*b*) *Myxœdema Proper.*—In this, women are very much more frequently affected than men—in a ratio of one to six. The disease may affect several members of a family, and it may be transmitted through the mother. In some instances there has been first the appearance of exophthalmic goitre. Though occurring most commonly in women, it seems to have no special relation to the catamenia or to pregnancy, though in one instance the symptoms of myxœdema disappeared during pregnancy. It is not so common in this country as in England. The symptoms of this form, as given by Ord,* are marked increase in the general bulk of the body, a firm, inelastic swelling of the skin, which does not pit on pressure, dryness and roughness, which tend, with the swelling, to obliterate in the face the lines of expression, imperfect nutrition of the hair, local tumefaction of the skin and subcutaneous tissues, particularly in the supraclavicular region. The physiognomy is altered in a remarkable way, the features are coarse and broad, the lips thick, the nostrils broad and thick, and the mouth is enlarged. Over the cheeks, sometimes the nose, there is a reddish patch. There is a striking slowness of thought and of movement. The memory becomes defective, the patients become irritable and suspicious, and there may be headache. In some instances there are delusions and hallucinations, leading to a final condition of dementia. The gait is heavy and slow. The temperature may be below normal. The functions of the heart, lungs, and abdominal organs are normal. Hæmorrhage sometimes occurs. Albuminuria is sometimes present, more rarely glycosuria. Death is usually due to some intercurrent disease. The thyroid gland is diminished in size and may become completely atrophied and converted into a fibrous mass. The subcutaneous fat is abundant and in one or two instances a great increase in the mucin has been found.

The course of the disease is slow but progressive, and extends over ten or fifteen years. I have recently had under observation a case to which the term acute myxœdema might be applied. A young man, aged twenty, presented a gradual enlargement of the face, particularly of the lips and cheeks and nose, without actual œdema. The backs of the hands were also swollen, but did not pit. The condition came on with enlargement of the thyroid, and, after persisting for between three and four months, is now gradually subsiding.

(*c*) *Operative Myxœdema; Cachexia Strumipriva.*—Horsley, in a series of interesting experiments, showed that complete removal of the thyroid in monkeys was followed by the production of a condition similar to that of myxœdema and often associated with spasms or tetanoid contractures, and followed by apathy and coma. When the monkeys were kept warm myxœdema was averted, and, instead of an acute myxœdema, the animals devel-

* Report on Myxœdema, Clinical Society's Transactions, 1888.

oped a condition which closely resembled cretinism. An identical condition may follow extirpation of the thyroid in man. Kocher, of Bern, found that after complete extirpation a cachectic condition followed in many cases, the symptoms of which are practically identical with those of myxœdema. The disease follows only a certain number of total and a much smaller proportion of partial removals of the thyroid gland. Of 408 cases, in 69 the operative myxœdema developed. It has been thought that if a small fragment of the thyroid remains, or if there are accessory glands, which in animals are very common, these symptoms do not develop. It is possible that in men, in the cases of complete removal, the accessory fragments subserve the function of the gland. Operative myxœdema is very rare in this country; the only case of which I know is a patient of McGraw's, of Detroit.

It is evident that the thyroid gland supplies some essential secretion of first importance to normal metabolism. What this is or how it acts is at present beyond our knowledge.

The *diagnosis* of the disease is easy. Bright's disease is the only condition for which it could be readily mistaken, but the absence of pitting, the curious condition of the face, and the absence of albumen in the urine are features which would readily distinguish it.

Unfortunately, no satisfactory treatment is known. The patients suffer in cold and improve greatly in warm weather. They should, therefore, be kept at an even temperature, and should, if possible, move to a warm climate during the winter months.

SECTION VII.

DISEASES OF THE KIDNEYS.

I. ANOMALIES IN FORM AND POSITION.

Anomalies in Form.—These rarely come within the scope of the physician. Atrophy or congenital absence of one kidney is associated with great enlargement of the other organ. Fused kidneys may have a horseshoe shape, or both organs may form a large mass, which is often displaced, being either in an iliac fossa or in the middle line of the abdomen, or even in the pelvis. Under these circumstances it may be mistaken for a new growth. In Polk's case the organ was removed under the belief that it was a floating kidney.* The patient lived eleven days, had complete anuria, and it was found post mortem that a single unsymmetrical kidney, as this form is called, had been removed.

Movable Kidney

(Floating Kidney; Palpable Kidney; Ren mobilis; Nephroptosis).

The kidney is held in position by its fatty capsule, by the peritonæum which passes in front of it, and by the blood-vessels. The lower edge of the left kidney is nearly two inches from the iliac crest, a little below the level of the second lumbar spine; that of the right is usually from one half to three quarters of an inch lower. Normally the kidney is firmly fixed, but under certain circumstances one or another organ, more rarely both, becomes movable. In rare cases the kidney is surrounded, to a greater or less extent, by the peritonæum, and is anchored at the hilus by a mesonephron. Some would limit the term floating kidney to this condition.

Movable kidney is almost always acquired. It is most common in women. Of the 667 cases collected in the literature by Kuttner, 584 were in women and only 83 in men. It is more common on the right than on the left side. Of 727 cases analyzed by this author, it occurred on the right in 553 cases, on the left in 81, and on both sides in 93. The

* New York Medical Journal, 1883.

greater frequency of the condition in women may be attributed to compression of the lower thoracic zone by tight lacing, and, more important still, to the relaxation of the abdominal walls which follows repeated pregnancies. This does not account for all the cases, as movable kidney is by no means uncommon in nulliparæ. Drummond believes that in a majority of the cases there is a congenitally relaxed condition of the peritoneal attachments. Wasting of the fat about the kidney may be a cause in some instances. Trauma and the lifting of heavy weights are occasionally factors in its production. The kidney is sometimes dragged down by tumors. The greater frequency on the right side is probably associated with the position of the kidney just beneath the liver, and the depression to which the organ is subjected with each descent of the diaphragm in inspiration.

And, lastly, movable kidney is met with in many cases which present that combination of neurasthenia with gastro-intestinal disturbance which has been described by Glénard * as *enteroptosis*.

To determine the presence of a movable kidney the patient should be placed in the dorsal position, with the head moderately low and the abdominal walls relaxed. The left hand is placed in the lumbar region behind the eleventh and twelfth ribs; the right hand in the hypochondriac region, in the nipple line, just under the edge of the liver. Bimanual palpation may detect the presence of a firm, rounded body just below the edge of the ribs. If nothing can be felt the patient should be asked to draw a deep breath, when, if the organ is palpable, it is touched by the fingers of the right hand. Various grades of mobility may be recognized. It may be possible barely to feel the lower edge on deep palpation—*palpable kidney*—or the organ may be so far displaced that on drawing the deepest breath the fingers of the right hand may be in a thin person slipped above the upper end of the organ, which can be readily held down, but cannot be pushed below the level of the navel—*movable kidney*. In a third group of cases the organ is freely movable, and may even be felt just above Poupart's ligament, or may be in the middle line of the abdomen, or can even be pushed over beyond this point. To this the term *floating kidney* is appropriate, whether the organ has a mesonephron or not.

And, lastly, a dislocated kidney may become fixed in an abnormal position. This is extremely rare, and in a very large number of cases I have found only one instance of the kind. A woman, aged twenty-nine, with four children, had nervous symptoms with abdominal pain, and had been much worried by the discovery of a tumor, just to the right of the middle line, close to the navel. It was not movable, but the distinctly reniform shape and the depression at the left margin indicated that it was doubtless a dislocated kidney which had become fixed.

* Revue de Médécine, 1887; Pourcelot, Paris Thesis, 1889.

The movable kidney is not painful on pressure, except when it is grasped very firmly, when there is a dull pain, or sometimes a sickening sensation. Examination of the patient from behind may show a distinct flattening in the lumbar region on the side in which the kidney is mobile.

Symptoms.—In a large majority of cases the condition gives no trouble, and it is well, if detected accidentally, not to let the patient know of its presence. In other instances there is pain in the lumbar region or a sense of dragging and discomfort, or there may be intercostal neuralgia. In a large group the symptoms are those of neurasthenia with dyspeptic disturbance. In women the hysterical symptoms may be marked, and in men various grades of hypochondriasis. The gastric disturbance is usually a form of nervous dyspepsia. Dilatation of the stomach has been observed, owing, as suggested by Bartels, to pressure of the dislocated kidney upon the duodenum. This view has been supported by Oser, Landau, and Ewald. On the other hand, Litten holds that the dilatation of the stomach is the cause of the mobility of the kidney, and he found in 40 cases of depression and dilatation of the stomach 22 instances of dislocation of the kidney on the right side. My own experience coincides with that of Drummond, who has very exceptionally found the two conditions to coexist. While not denying the possibility of causal relationship between the two, it seems probable, considering the frequency of floating kidney, that the complication is only a coincidence. The association, however, with a *depressed* stomach is certainly not uncommon in women. Constipation is not infrequent. Some writers have described pressure upon the gall-ducts, with jaundice, but it is not very likely to occur.

Under the name *enteroptosis*, Glénard has described a special symptom group characterized by nervous dyspepsia, prolapse of the abdominal organs, particularly the transverse colon, with looseness of the mesenteric and peritoneal attachments, so that there is a falling down of the viscera (splanchnoptosis). Dilatation of the stomach and mobility of the kidney are very commonly associated with this state. Glénard held that he could feel the prolapsed transverse colon as a narrow band, but Ewald states correctly that this is the pancreas, which in many of these cases can be distinctly palpated. According to Glénard, the kink in the colon causes the constipation, while the depression of the stomach and intestines leads to vascular disturbance and impairment of the motor and secretory functions.

In floating kidney there are attacks (simulating gastralgia or renal colic) characterized by severe abdominal pain, chills, nausea, vomiting, fever, and collapse. Scarcely any mention is made of such symptoms, which were first described by Dietl in 1864, and a more wide-spread knowledge of their occurrence in connection with this condition is desirable. My attention was called to them in 1880 by Palmer Howard in the case of a stout lady, who suffered repeatedly with the most severe attacks of abdominal pain and vomiting, which constantly required morphia. A

tumor was discovered a little to the right of the navel, and the diagnosis of probable neoplasm was concurred in by Flint (Sr.) and Gaillard Thomas. The patient lost weight rapidly, became emaciated, and in the spring of 1881 again went to New York, where she saw Van Buren, who diagnosed a floating kidney and said that these paroxysms were associated with it in a gouty person. He cut off all stimulants, reassured the lady that she had no cancer, and from that time she rapidly recovered, and the attacks have been few and far between. In this patient any over-indulgence in eating or in drinking is still liable to be followed by a very severe attack. These attacks may also be mistaken for renal colic, and the operation of nephrotomy has been performed.

In other instances the attacks of pain may be thought to be due to intestinal disease or to recurring appendicitis. The cause of these paroxysmal attacks is not quite clear. Dietl thought they were due to strangulation of the kidney or to twists or kinks in the renal vessels due to the extreme mobility. During the attacks the urine is sometimes high-colored and contains an excess of uric acid or of the oxalates. It is stated, too, that blood or pus may be present. The kidney may be tender, swollen, and less freely movable. Intermittent hydronephrosis has sometimes been associated with movable kidney.

The *diagnosis* is rarely doubtful, as the shape of the organ is usually distinctive and the mobility marked. Tumors of the gall-bladder, ovarian growths, and tumors of the bowels may in rare instances be confounded with it.

Treatment.—The kidney has been extirpated in many instances, but the operation is not without risk, and there have been several fatal cases. Stitching of the kidney—nephrorrhaphy—as recommended by Hahn, is the most suitable procedure, and statistics recently published by Keen show that relief is afforded in many cases by the procedure. It does not, however, always succeed.

The treatment by trusses and bandages is not satisfactory, though great relief is sometimes obtained. As a rule, bandages, with pads pressing to the right of the navel, are not well borne, as the kidney is often sensitive. In some instances, however, the greatest relief is experienced by this procedure. An air-pad beneath the bandage, as recommended by Newman, is probably the best. In other cases a broad bandage well padded in the lower abdominal zone pushes up the intestines and makes them act as a support. In the attacks of severe colic morphia is required. When dependent, as seems sometimes the case, upon an excess of uric acid or the oxalates, the diet must be carefully regulated.

II. CIRCULATORY DISTURBANCES.

Normally the secretion of urine is accomplished by the maintenance of a certain blood-pressure within the glomeruli and by the activity of the renal epithelium. Bowman's views on this question have been generally accepted, and the watery elements are held to be filtered from the glomeruli; the amount depending on the rapidity and the pressure of the blood current; the quality, whether normal or abnormal, depending upon the integrity of the capillary and glomerular epithelium; while the greater portion of the solid ingredients are excreted by the epithelium of the convoluted tubules. The integrity of the epithelium covering the capillary tufts within Bowman's capsule is essential to the production of a normal urine. If under any circumstances their nutrition fails, as when, for example, the rapidity of the blood-current is lowered, so that they are deprived of the necessary amount of oxygen, the material which filters through is no longer normal (i. e., water), but contains serum albumen. Cohnheim has shown that the renal epithelium is extremely sensitive to circulatory changes, and that compression of the renal artery for only a few minutes causes serious disturbance.

The circulation of the kidney is remarkably influenced by reflex stimuli coming from the skin. Exposure to cold causes heightened blood-pressure within the kidneys and increased secretion of urine. So also in the chills of malaria, after which a large amount of pale urine may be passed.

Congestion of the Kidneys.—(1) *Active Congestion; Hyperæmia.*—Acute congestion of the kidney is met with in the early stage of nephritis, whether due to cold or to the action of poisons and severe irritants. Turpentine, cubebs, cantharides, and copaiba are all stated to cause extreme hyperæmia of the organ. The most typical congestion of the kidney which we see post mortem is that in the early stage of acute Bright's disease, when the organ may be large, soft, of a dark color, and on section blood drips from it freely.

It has been held that in all the acute fevers the kidneys are congested, and that this explained the scanty, high-colored, and often albuminous urine. On the other hand, by Roy's oncometer, Walter Mendelson has shown that the kidney in acute fever is in a state of extreme anæmia, small, pale, and bloodless; and that this anæmia, increasing with the pyrexia and interfering with the nutrition of the glomerular epithelium, accounts for the scanty, dark-colored urine of fever and for the presence of albumen. In the prolonged fevers, however, it is probable that relaxation of the arteries again takes place. Certainly it is rare to find post mortem such a condition of the kidney as is described by Mendelson. On the contrary, the kidney of fever is commonly swollen, the blood-vessels are congested, and the cortex frequently shows traces of cloudy swelling. However, the circulatory disturbances in acute fevers are probably less im-

portant than the irritative effects of either the specific agents of the disease or the products produced in their growth, or in the altered metabolism of the tissues. The urine is diminished in amount, and may contain albumen and tube-casts.

(2) *Passive Congestion; Mechanical Hyperæmia.*—This is found in cases of chronic disease of the heart or lung, with impeded circulation, and as a result of pressure upon the renal veins by tumors, the pregnant uterus, or ascitic fluid In the cardiac kidney, as it is called, the cyanotic induration associated with chronic heart-disease, the organs are enlarged and firm, the capsule strips off, as a rule, readily, the cortex is of a deep red color, and the pyramids of a purple red. The section is coarse-looking, the substance is very firm, and resists cutting and tearing. The interstitial tissue is increased, and there is a small celled infiltration between the tubules. Here and there the Malpighian tufts have become sclerosed. The blood-vessels are usually thickened, and there may be more or less granular, fatty, or hyaline changes in the epithelium of the tubules. The condition is indeed a diffuse nephritis. The urine is usually reduced, is of high specific gravity, and contains more or less albumen. Hyaline tube-casts and blood-corpuscles are not uncommon. In uncomplicated cases of the cyanotic induration uræmia is rare. On the other hand, in the cardiac cases with extensive arterio-sclerosis, the kidneys are more involved and the renal function is likely to be disturbed.

III. ANOMALIES OF THE URINARY SECRETION.

1. Hæmaturia.

The following division may be made of the causes of hæmaturia:

(1) *General Diseases.*—The malignant forms of the acute specific fevers, such as small-pox, malaria, yellow fever, etc.; scurvy, purpura, and hæmophilia. Occasionally in leukæmia hæmaturia occurs.

(2) *Renal Causes.*—Acute congestion and inflammation, as in Bright's disease, or the effect of toxic agents, such as turpentine, carbolic acid, and cantharides. When the carbolic spray was in use many surgeons suffered from hæmaturia in consequence of this poison. Renal infarction, as in ulcerative endocarditis. New growths, in which the bleeding is usually profuse. Tubercle rarely causes hæmaturia, though at the onset, when the papillæ are involved, there may be bleeding. Stone in the kidney is a frequent cause. Parasites: The *filaria sanguinis hominis* and the *Bilharzia* cause a form of hæmaturia met with in the tropics. The echinococcus is rarely associated with hæmorrhage.

(3) *Affections of the Urinary Passages.*—Stone in the ureter, malignant disease or ulceration of the bladder, the presence of a calculus, parasites, and, very rarely, ruptured veins in the bladder. Bleeding from the

urethra occasionally occurs in gonorrhœa and as a result of the lodgment of a calculus.

(4) *Traumatism.*—Injuries may produce bleeding from any part of the urinary passages. By a fall or blow on the back the kidney may be ruptured, and this may be followed by very free bleeding; less commonly the blood comes from injury of the bladder or of the prostate. Blood from the urethra is frequently due to injury by the passage of a catheter, or sometimes to falls or blows.

And, lastly, there are cases in which hæmaturia occurs for a long time without discoverable cause, particularly in young persons. The health may not be seriously impaired. Gull has characterized, in a happy way, a case of this kind as one of renal epistaxis.

Of special interest is the malarial hæmaturia which prevails in certain districts and has already been considered in the section on paludism.

The *diagnosis* of hæmaturia is usually easy. The color of the urine varies from a light smoky to a bright red, or it may have a dark porter color. Examined with the microscope, the blood-corpuscles are readily recognized, either plainly visible and retaining their color, in which case they are usually crenated, or simply as shadows. In ammoniacal urine or urines of low specific gravity the hæmoglobin is rapidly dissolved from the corpuscles, but in normal urine they remain for many hours unchanged.

Other tests are rarely necessary. The guaiacum test consists of the addition to the urine, in a test-tube, of a drop or two of the tincture of guaiacum and two minims of ozonic ether. A blue color forms at the line of contact of the two fluids and diffuses itself through the ether.

The spectroscopical examination of the urine may show either the single band of reduced hæmoglobin or the double band of oxyhæmoglobin between the lines D and E.

It is important to distinguish between blood coming from the bladder and from the kidneys, though this is not always easy. From the bladder the blood may be found only with the last portions of urine, or only at the termination of micturition. In hæmorrhage from the kidneys, the blood and urine are intimately mixed. Clots are more commonly found in the blood from the kidneys, and may form moulds of the pelvis or of the ureter. When the seat of the bleeding is in the bladder, on washing out this organ, the water is more or less blood-tinged; but if the source of the bleeding is higher, the water comes away clear. In many instances it is difficult to settle the question by the examination of the urine alone, and the symptoms and the physical signs must also be taken into account.

2. Hæmoglobinuria.

This condition is characterized by the presence of blood-pigment in the urine. The blood-cells are either absent or in insignificant numbers. The coloring matter is not hæmatin, as indicated by the old name, *hæma-*

tinuria, nor in reality always hæmoglobin, but it is most frequently methæmoglobin. The urine has a red or brownish-red, sometimes quite black color, and usually deposits a very heavy brownish sediment. When the hæmoglobin occurs only in small quantities, it may give a lake or smoky color to the urine. Microscopical examination shows the presence of granular pigment, sometimes fragments of blood-disks, epithelium, and very often darkly pigmented urates. The urine is also albuminous. The number of red blood-corpuscles bears no proportion whatever to the intensity of the color of the urine. Examined spectroscopically, there are either the two absorption bands of oxyhæmoglobin, which is rare, or, more commonly, there are the three absorption bands of methæmoglobin, of which the one in the red near *C* is characteristic. Two clinical groups may be distinguished.

(1) **Toxic Hæmoglobinuria.**—This is caused by poisons which produce rapid dissolution of the blood-corpuscles, such as chlorate of potash in large doses, pyrogallic acid, carbolic acid, arseniuretted hydrogen, carbon dioxide, naphthol, and muscarine; also the poisons of scarlet fever, yellow fever, typhoid fever, malaria, and syphilis. It has also followed severe burns. Exposure to excessive cold and violent muscular exertion are stated to produce hæmoglobinuria. A most remarkable toxic form occurs in horses, coming on with great suddenness and associated with paresis of the hind legs. Death may occur in a few hours or a few days. Horses are attacked only after being stalled for some days and then taken out and driven, particularly in cold weather. The affection is common in horses in this country. The form of hæmoglobinuria from cold and exertion is extremely rare No instance of it, even in association with frost-bites, came under my observation in Canada. Blood transfused from one mammal into another causes dissolution of the corpuscles with the production of hæmoglobinuria; and, lastly, there is the *epidemic hæmoglobinuria* of the new-born, associated with jaundice, cyanosis, and nervous symptoms.

(2) **Paroxysmal Hæmoglobinuria.**—This rare disease is characterized by the occasional passage of bloody urine, in which the coloring matter only is present. It is more frequent in males than in females, and occurs chiefly in adults. It seems specially associated with cold and exertion, and has often been brought on, in a susceptible person, by the use of a cold foot-bath. Paroxysmal hæmoglobinuria has been found, too, in persons subject to the various forms of Raynaud's disease. Many regard the relation between these two affections as extremely close; some hold that they are manifestations of one and the same disorder. Druitt, the author of the well-known Surgical Vade-mecum, has given a graphic description of his sufferings, which lasted for many years, and were accompanied with local asphyxia and local syncope. The connection, however, is not very common. In only one of the cases of Raynaud's disease which I have seen was paroxysmal hæmoglobinuria present, and in it epileptic attacks occurred at the same time. The relation of the disease to

malaria is not so close as has been thought by many writers. No doubt it has been frequently confounded with a malarial hæmaturia. The attacks may come on suddenly after exposure to cold or as a result of mental or bodily exhaustion. They may be preceded by chills and pyrexia. In other instances the temperature is subnormal. There may be vomiting and diarrhœa. Pain in the lumbar region is not uncommon. The hæmoglobinuria rarely persists for more than a day or two—sometimes, indeed, not for a day. There are instances in which, even in the course of a single day, there have been two or three paroxysms, and in the intervals clear urine has been passed. Jaundice has been present in a number of cases. According to Ralfe, paroxysmal hæmoglobinuria may alternate with general symptoms of the same character, but associated only with the passage of albumen and an increased quantity of urea in the urine. In such cases he supposes that the toxic agent, whatever its nature, has destroyed only a limited number of the corpuscles, the coloring matter of which is readily dealt with by the spleen and liver, while the globulin is excreted in the urine. The cases are rarely if ever fatal.

The essential pathology of the disease is unknown, and it is difficult to form a theory which will meet all the facts—particularly the relation with Raynaud's disease, which is rightly regarded as a vaso-motor disorder. Increased hæmolysis and dissolution of the hæmoglobin in the blood-serum (hæmoglobinæmia) precedes, in each instance, the appearance of the coloring matter in the urine; but, as Ponfick has shown, the amount of free hæmoglobin must reach a certain grade before it is excreted.

Treatment.—In all forms of hæmaturia rest is essential. In that produced by renal calculi the recumbent posture may suffice to check the bleeding. Full doses of acetate of lead and opium should be tried, then ergot, gallic and tannic acid, and the dilute sulphuric acid. The oil of turpentine, which is sometimes recommended, is a risky remedy in hæmaturia. Extr. hamamelis virgin. and extr. hydrastis canad. are also recommended. Cold may be applied to the loins or dry cups in the lumbar region.

The treatment of hæmoglobinuria is unsatisfactory. Nothing seems to check the occurrence of the attacks. During the paroxysm the patient should be kept warm and given hot drinks. Quinine is recommended in large doses, on the supposition—as yet unwarranted—that the disease is specially connected with malaria. If there is a syphilitic history iodide of potassium, in full doses, may be tried. In a warm climate the attacks are much less frequent.

III. Albuminuria.

The presence of albumen in the urine, formerly regarded as indicative of Bright's disease, is now recognized as occurring under many circumstances without the existence of serious organic change in the kidney.

Two groups of cases may be recognized—those in which the kidneys show no coarse lesions, and those in which there are evident anatomical changes.

Albuminuria without Coarse Renal Lesions.—(*a*) *Functional, So-called Physiological, Albuminuria.*—In a normal condition of the kidney only the water and the salts are allowed to pass from the blood. When albuminous substances transude there is probably disturbance in the nutrition of the epithelium of the capillaries of the tuft, or of the cells surrounding the glomerulus. This statement is still, however, in dispute, and Senator, Grainger Stewart, and others hold that there is a physiological albuminuria which may follow muscular work, the ingestion of food rich in albumen, violent emotions, cold bathing, and dyspepsia. The differences of opinion on this point are striking, and observers of equal thoroughness and reliability have arrived at directly opposite conclusions. The presence of albumen in the urine, in any form and under any circumstance, may be regarded as indicative of change in the renal or glomerular epithelium, a change, however, which may be transient, slight, and unimportant, depending upon variations in the circulation or upon the irritating effects of substances taken with the food or temporarily present, as in febrile states.

Much attention has been given of late years to the albuminuria of adolescence, or *cyclic albuminuria*, which is also believed to be a functional disorder. A majority of the cases occur in young persons—boys more commonly than girls—and the condition is often discovered accidentally. The urine, as a rule, contains only a very small quantity of albumen, but in some instances large quantities are present. The most striking feature is the variability. It may be absent in the morning and only present after exertion, or it may be greatly increased after taking food, particularly proteids. The quantity of urine may be but little if at all increased, the specific gravity is usually normal, and the color may be high. Occasionally, hyaline casts may be found, and in some instances there has been transient glycosuria. As a rule, the pulse is not of high tension and the second aortic sound is not accentuated.

Various forms of this affection have been recognized by writers, such as neurotic, dietetic, cyclic, intermittent, and paroxysmal—names which indicate the characters of the different varieties. A large proportion of the cases get well after the condition has persisted for a variable period. This in itself is an evidence that the changes, whatever their nature, were transient and slight. In these instances the albumen exists in small quantity, tube-casts are not present, and the arterial tension is not increased. In a second group the albumen is more persistent, the amount is larger, though it may vary from day to day, and the pulse tension is increased. In such instances the persistent albuminuria probably indicates actual organic change in the kidney.

(*b*) *Febrile Albuminuria.*—Pyrexia, by whatever cause produced,

may cause slight albuminuria. The presence of the albumen is due to slight changes in the glomeruli induced by the fever, such as cloudy swelling, which cannot be regarded as an organic lesion. It is extremely common, occurring in pneumonia, diphtheria, typhoid fever, and even in the fever of acute tonsillitis. The amount of albumen is slight, and it usually disappears from the urine with the cessation of the fever.

(*c*) *Hæmic Changes.*—Purpura, scurvy, chronic poisoning by lead or mercury, syphilis, leukæmia, and profound anæmia may be associated with slight albuminuria. Abnormal ingredients in the blood, such as bile-pigment and sugar, may cause the passage of small amounts of albumen.

The transient albuminuria of pregnancy may belong to this hæmic group, although in a majority of such cases there are changes in the renal tissue. Albumen may be found sometimes after the inhalation of ether or chloroform.

(*d*) Albuminuria occurs in certain affections of the *nervous system.* This so-called neurotic albuminuria is seen after an epileptic seizure and in apoplexy, tetanus, exophthalmic goitre, and injuries of the head.

Albuminuria with Definite Lesions of the Urinary Organs.—(*a*) Congestion of the kidney, either active, such as follows exposure to cold and is associated with the early stages of nephritis, or passive, due to obstructed outflow in disease of the heart or lungs, or to pressure on the renal veins by the pregnant uterus or tumors.

(*b*) Organic disease of the kidneys—acute and chronic Bright's disease, amyloid and fatty degeneration, suppurative nephritis, and tumors.

(*c*) Affections of the pelvis, ureters, and bladder, when associated with the formation of pus.

Tests for Albumen.—Both morning and evening urine should be examined, and in doubtful cases at least three specimens. If turbid, the urine should be filtered, though turbidity from the urates is of no moment, since it disappears at once on the application of heat.

Heat and Nitric-acid Test.—The urine is boiled in a test-tube over a spirit-lamp, and a drop of nitric acid is then added. If a cloudiness occurs on boiling, it may be due to phosphates, which are dissolved on the addition of an acid. Persistence of the cloudiness indicates albumen.

Heller's Test.—A small quantity of fuming nitric acid is poured into the test-tube, and with a pipette the urine is allowed to flow gently down the side upon the acid. At the line of junction of the two fluids, if albumen is present, a white ring is formed. This contact method is trustworthy, and, for the routine clinical work, is probably the most satisfactory. A diffused haze, due to mucin, is sometimes seen just above the white ring of albumen. A colored ring at the junction of the acid and the urine is due to the oxidation of the coloring matters in the urine.

Sir William Roberts strongly recommends the *magnesium-nitric test.* One volume of strong nitric acid is mixed with five volumes of the saturated

solution of sulphate of magnesium. This is used in the same way as the nitric acid in Heller's test.

Picric acid, introduced by George Johnson, is a delicate and useful test for albumen. A saturated solution is used and employed as in the contact method. It has been urged against this test that it throws down the mucin, peptones, and certain vegetable alkaloids, but these are dissolved by heat.

For minute traces of albumen the trichloracetic acid may be used, or Millard's fluid, which is extremely delicate and consists of glacial carbolic acid (ninety-five per cent), 2 drachms; pure acetic acid, 7 drachms; liquor potassæ, 2 ounces 6 drachms.

A quantitative estimate of the albumen can be made by means of Esbach's tube, but the rough method of heating and boiling a certain quantity of acidulated urine in a test-tube and allowing it to stand, is often employed. The depth of deposit can then be compared with the whole amount of urine, and the proportion is expressed as a mere trace, almost solid, one fourth, one half, and so on. This, of course, does not give an accurate indication of the proportion of albumen in the total quantity of urine. For the more elaborate methods the reader is referred to the works on urinalysis.

The above tests refer entirely to serum albumen. Other albuminous substances occur, such as serum globulin, peptones, and hemialbumose. By saturating the urine with magnesium sulphate, the globulin is precipitated, coagulated, and then readily separated from the serum albumen.

Traces of *peptones* are found in the urine in many acute diseases and in chronic suppuration. They are not precipitated by heat or nitric acid, but are thrown down by picric acid and dissolved by heat. If the urine contains peptones, a rose or pinkish tint is formed at the junction of the two fluids when urine is allowed to flow gently into a test-tube containing Fehling's solution. Peptonuria has no clinical significance.

Propepton, or hemialbumose, is not of any practical importance. It was found by Bence-Jones in the urine in osteomalacia, and occurs occasionally in other affections.

Prognosis.—This depends, of course, entirely upon the cause. Febrile albuminuria is transient, and in a majority of the cases depending upon hæmic causes the condition disappears and leaves the kidneys intact. An occasional trace of albumen in a man over forty, with or without a few hyaline casts, and with increased tension and thick vessel walls, usually indicates changes in the kidneys. The persistence of a slight amount of albumen in young men without increased arterial tension is less serious as even after continuing for years it may disappear. I have already spoken of the outlook in the so-called cyclic albuminuria.

Practically in all cases the presence of albumen indicates a change of some sort in the glomeruli, the nature, extent, and gravity of which it is difficult to estimate, so that other considerations, such as the presence of

tube-casts, the existence of increased tension, the general condition of the patient, and the influence of digestion upon the albumen, must be carefully considered.

The physician is daily consulted as to the relation of albuminuria and life assurance. As his function is to protect the interests of the company, he should reject all cases in which albumen occurs in the urine. It is even doubtful if an exception should be made in young persons with transient albuminuria. Naturally, companies lay great stress upon the presence or absence of albumen, but in the most serious and fatal malady with which they have to deal, chronic interstitial nephritis, the albumen is often absent or transient, even when the disease is well developed. After the fortieth year, from a standpoint of life insurance, the state of the arteries is far more important than the condition of the urine.

IV. Pyuria (*Pus in the Urine*).

Causes.—(1) *Pyelitis and Pyelonephritis.*—In large abscesses of the kidney, pyonephrosis, the pus may be intermittent, and for days or even weeks the urine is free. In calculous and tuberculous pyelitis the pyuria is usually continuous, though varying in intensity. In these cases, as a rule, the pus is mixed with the urine, which is acid in reaction. In the early stages of pyelitis the transitional epithelium may be abundant, but is not in any way distinctive. In the pyelitis and pyelonephritis following cystitis the urine is usually alkaline, and contains more mucus; micturition is usually more frequent, and the history points to a previous bladder affection.

(2) *Cystitis.*—The urine is alkaline, often fetid, the pus ropy, and the amount of urine greatly increased. The ropy, thick mucus usually comes with the last portions of the urine. Triple phosphate crystals may be present in the freshly passed urine.

(3) *Urethritis*, particularly gonorrhœa. The pus appears first, is in small quantities, and there are signs of local inflammation.

(4) In *leucorrhœa* the quantity of pus is usually small, and large flakes of vaginal epithelium are numerous. In doubtful cases, when leucorrhœa is present, the urine should be withdrawn by a catheter.

(5) *Rupture of Abscesses into the Urinary Passages.*—In such cases as pelvic or perityphlitic abscess there have been previous symptoms of pus formation. A large amount is passed within a short time, then the discharge stops abruptly or rapidly diminishes within a few days.

Pus gives to the urine a white or yellowish-white appearance. On settling there is a heavy grayish sediment, and the supernatant fluid is usually turbid. The sediment is often tenacious and ropy. The reaction is generally alkaline, and the odor may be ammoniacal even when passed. Examination with the microscope reveals the presence of a large number of pus-corpuscles, which are usually, when the pus comes from the blad-

der, well formed; the protoplasm is granular, and often shows many translucent processes.

The only sediment likely to be confounded with pus is that of the phosphates; but it is whiter and less dense, and is distinguished immediately by microscopical examination.

With the pus there is always more or less epithelium from the bladder and pelvis, but since in these situations the forms of cells are practically identical, they afford no information as to the locality from which the pus has come.

The treatment of pus in the urine is considered under the conditions in which it occurs.

V. Chyluria—Non-parasitic.

This is a rare affection, occurring in temperate regions and unassociated with the *filaria hominis sanguinis*. The urine is of an opaque white color; it resembles milk closely, is occasionally mixed with blood (hæmatochyluria), and sometimes coagulates into a firm, jelly-like mass. In other instances there is at the bottom of the vessel a loose clot which may be distinctly blood-tinged. Under the microscope the turbidity seems to be caused by numerous minute granules—more rarely oil droplets similar to those of milk. Traces of albumen are usually present. The amount of urine passed is generally increased, and the chylous condition is intermittent. It may persist for years without deterioration of health or evidence of serious disease.

Since the discovery of the *filaria hominis sanguinis* it has been incorrectly held by some that all of the cases of chyluria are of this parasitic nature. I had an opportunity in Montreal of making a careful study of a French-Canadian woman, a patient of J. B. McConnell's, who had had chyluria for more than thirteen years. The urine was quite milky in color and occasionally mixed with blood. Neither ova nor embryos were found in the urine or in the blood examined at night. After her death I was enabled to make a thorough dissection of the abdominal lymph vessels, which were found perfectly normal. The thoracic duct was not enlarged, the renal lymphatics were not distended; the kidneys were increased in size, but showed no special changes. The most careful examination of the lymph glands and vessels failed to reveal the presence of parasites.

The pathology of the condition is unknown. No known remedies have any influence upon the chyluria.

(For parasitic chyluria see Filariasis.)

VI. Lithuria (*Lithæmia; Lithic-acid Diathesis*).

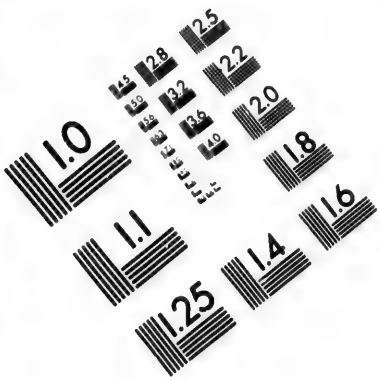
The amount of uric acid excreted daily depends greatly upon the diet, ranging from half a gramme on a vegetable to as high, even, as two

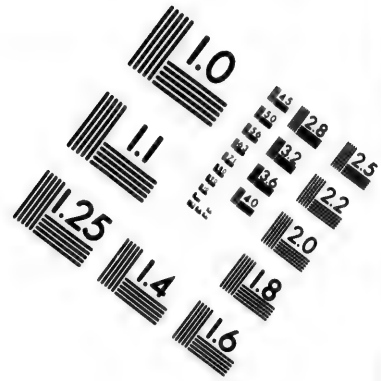
grammes on an animal diet. In the urine of herbivora it occurs only in traces. In that of carnivora it may be absent altogether. On the other hand, in the urine of birds and reptiles it is the chief nitrogenous ingredient. As Sir William Roberts remarks, its presence in the human urine is somewhat of an anomaly, as its place is very much better taken by urea, which is easily soluble and better adapted to the mammalian plan of a liquid urine. He regards it as a sort of vestigial remnant.

Place and Mode of Formation of the Uric Acid.—It is now very generally conceded that uric acid is formed in the tissues and excreted by the kidneys. It may occur in traces in the blood even in health. Von Jaksch, who has recently examined the blood of 109 individuals, found no trace in 9 healthy persons, nor was it present in cases of typhoid fever or in nervous affections or in diseases of the liver and gastro-intestinal canal, except when anæmia coexisted. On the other hand, it was present in connection with all those diseased processes in which oxidation was disturbed, either directly, as in affections of the lungs, such as pneumonia, or indirectly, as in anæmia, in which the oxygen-carriers are deficient. According to Haig, the amount in the blood rises and falls with the degree of alkalinity (as more is held in solution), and all circumstances which increase this are associated with an increase in the amount of uric acid.

As to the place of formation, the experimental evidence points strongly to the liver, and, according to Minkowski, it is formed there by the synthesis of ammonia and lactic acid. The views, however, as to its place of production and the antecedents are by no means harmonious. Garrod still holds that the kidneys are concerned not only with its excretion, but with its formation. On the other hand, Ebstein thinks that it is chiefly produced in the muscles and in the bone marrow. Nor is it yet settled whether uric acid is only an intermediate step in the formation of urea or whether it has an independent origin.

Mode of Elimination.—Uric acid is extremely insoluble, a gramme requiring for its solution, at ordinary temperature, fourteen litres of water, and about half that amount at body temperature. In the 1,500 to 2,000 c. c. of urine passed in the day the uric acid could not be dissolved, but it is eliminated in combination as soluble salts, chiefly as urates of ammonium and sodium. The power in the blood of holding the uric acid in solution depends upon the degree of alkalinity; thus it has been long known that the excretion of uric acid some hours after breakfast is high. This is in what Sir William Roberts calls "the alkaline tide." Haig has shown that this excretion can be increased or diminished by increasing or diminishing the alkalinity of the blood; thus, under salicylate of soda, given in fifteen grain doses three times a day, the excretion of the uric acid is increased on the first and second days, and subsequently falls to the normal amount. He explains this by supposing that the salicylate finds a considerable quantity of uric acid stored in the liver, spleen, and other tissues, gets this into solution, and the greater part of it is passed in the urine. His obser-





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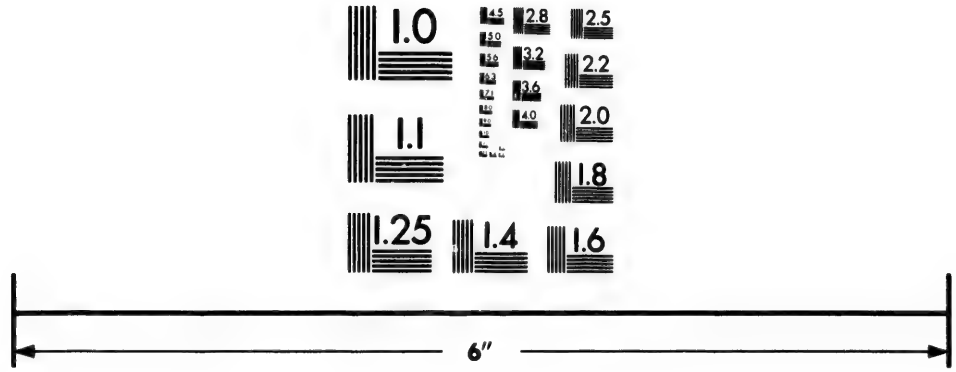

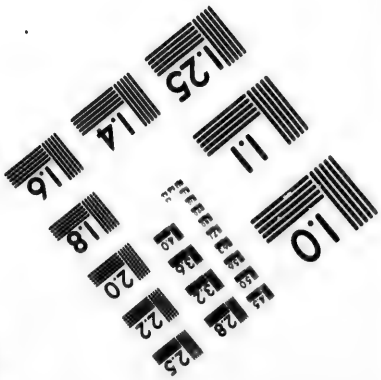

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vations indicate that alkalies, such as the phosphate of soda and compounds of salicylic acid, increase markedly the excretion of this ingredient in the urine, and also increase the amount of it in the blood, withdrawing it from the spleen and liver. On the other hand, acids, lead, and iron interfere with the solubility of the uric acid and with its elimination. A fact of great practical importance which he mentions is that lithia, "though a beautiful solvent of uric acid in a test-tube, yet when given to the human subject by mouth never reaches the uric acid at all, because it at once forms an insoluble compound with the phosphate of soda in the blood, thus removing from that fluid one of the natural solvents of uric acid, and diminishing its power of holding uric acid in solution." This is directly opposed to the prevalent ideas of the value of the lithia compounds in the uric-acid diathesis.

The pathology of uric acid is more a matter of defective elimination than of excessive formation. In conditions of the system associated with persistent diminished alkalinity of the blood the uric acid accumulates in the liver, spleen, and the joints. The degree of alkalinity of the kidney structure possibly, as Haig suggests, may have an influence in determining how much shall be excreted and how much retained, and, according to his views, it is this small remnant or overflow which accumulates in the blood and produces headache, high tension of the pulse, and mental depression, and when deposited in the joints causes gouty arthritis and the uratic lesions.

Occurrence in the Urine.—The uric acid occurs in combination chiefly with ammonium and sodium, forming the acid urates. In smaller quantities are the potassium, calcium, and lithium salts. The uric acid may be separated from its bases and crystallizes in rhombs or prisms, which are usually of a deep red color, owing to the staining of the urinary pigments. The sediment formed is granular and the groups of crystals look like grains of Cayenne pepper. It is very important not to mistake a deposit of uric acid for an excess. The deposition of numerous grains in the urine within a few hours after passing is more likely to be due to conditions which diminish the solvent power than to increase in the quantity. Of the conditions which cause precipitation of the uric acid Roberts gives the following: "(1) High acidity; (2) poverty in mineral salts; (3) low pigmentation; and (4) high percentage of uric acid." The grade of acidity is probably the most important element.

More common is the precipitation of amorphous urates, forming the so-called brick-dust or lateritious deposit, which has a pinkish color, due to the presence of urinary pigment. It is composed chiefly of the acid sodium urates. It occurs particularly in very acid urine of a high specific gravity. As the urates are more soluble in warm solutions they frequently deposit as the urine cools. Here, too, the deposition does not necessarily, indeed usually does not, mean an excessive excretion, but the existence of conditions favoring the deposit.

Murchison introduced the term lithæmia to designate certain symptoms due, as he supposed, to functional disturbance of the liver. Not only have his views been widely adopted, but, as is so often the case when we give the rein to theoretical conceptions of disease, the so-called manifestations of this state have so multiplied that some authors attribute to this cause a considerable proportion of the ailments affecting the various systems of the body. Thus one writer enumerates not fewer then thirty-nine separate morbid conditions associated with lithæmia. From what has been said as to our knowledge, or rather our lack of knowledge, of the mode of formation and elimination of uric acid it is very evident that the physiology of the subject must be widely extended before we are in a position to draw safe conclusions. Thus it is by no means sure that, as Murchison supposed, the essential defect is in a functional disorder of the liver, disturbing the metabolism of the albuminous ingredients, nor is it at all certain that the only offending substance is uric acid. Bouchard contends that the so-called lithiasis has little or nothing to do with disturbance in the function of the liver, and that it has not been shown that uric acid is the only or even the chief agent in producing the symptoms. In the present imperfect state of knowledge it is impossible with any clearness to define the pathology of the so-called uric-acid diathesis. We may say that certain symptoms arise in connection with defective food or tissue metabolism, more particularly of the nitrogenous elements. Deficient oxidation is probably the most essential factor in the process, with the result of the formation of less readily soluble and less readily eliminated products of retrograde metamorphosis. This faulty metabolism if long continued may lead to gout, with uratic deposits in the joints, acute inflammations, and arterial and renal disease. In a large group of cases the disturbed metabolism produces high tension in the arteries (probably as a direct sequence of interference with the capillary circulation) and ultimately degenerations in various tissues, particularly the scleroses.

Overeating and overdrinking, when combined with deficient muscular exercise, lie at the basis of this nutritional disturbance. The symptoms which are believed to characterize the uric-acid diathesis have already been briefly considered under the section on irregular gout, and the question of diet and exercise has also been there considered.

VII. Oxaluria.

Oxalic acid occurs in the urine, in combination with lime, forming an oxalate which is held in solution by the acid phosphate of soda. About .01 to .02 gramme is excreted in the day. It never forms a heavy deposit, but the crystals—usually octahedra, rarely dumb-bell-shaped—collect in the mucus-cloud and on the sides of the vessel. The amount varies extremely with the diet, and it is increased largely when such fruits and vegetables as tomatoes and rhubarb are taken. It is also a product of incomplete oxi-

dation of the organic substances in the body, and in conditions of increased metabolism the amount in the urine becomes larger. It is stated also to result from the acid fermentation of the mucus in the urinary passages and the crystals are usually abundant in spermatorrhœa.

When in excess and present for any considerable time, the condition is known as oxaluria, the chief interest of which is in the fact that the crystals may be deposited before the urine is voided, and form a calculus. It is held by many that there is a special diathesis associated with this state and manifested clinically by dyspepsia, particularly the nervous form, irritability, depression of spirits, lassitude, and sometimes marked hypochondriasis. There may be in addition neuralgic pains and the general symptoms of neurasthenia. The local and general symptoms are probably dependent upon some disturbance of metabolism of which the oxaluria is one of the manifestations. It is a feature also in many gouty persons, and in the condition called lithæmia.

VIII. Cystinuria.

Cystin does not occur in normal urine. It is very rarely met with, and its chief interest is owing to the fact that it may form a calculus. Its presence in the urine has been determined in many members of the same family, and the condition appears sometimes to be hereditary. As it contains sulphur, it is thought to be formed from the taurin of the bile.

IX. Phosphaturia.

The phosphoric acid is excreted from the body in combination with potassium, sodium, calcium, and magnesium, forming two classes, the alkaline phosphates of sodium and potassium and the earthy phosphates of lime and magnesia. "*The alkaline phosphates* exist in the blood in the form of neutral sodium and potassium phosphates (hydrogen-disodium phosphates, HNa_2PO_4), but appear in the urine as acid sodium and potassium phosphates (dihydrogen-sodium phosphates, H_2NaPO_4), and thus cause the acid reaction of that secretion. The change of the neutral into the acid salt is caused by the decomposition effected by the act of secretion, in which the bicarbonates and neutral phosphates in the blood change into carbonates and acid phosphates respectively." (Ralfe.)

Of the *earthy phosphates*, those of lime are abundant, of magnesium scanty. In urine which has undergone the ammoniacal fermentation, either inside or outside the body, there is in addition the ammonio-magnesium or triple phosphate, which occurs in triangular prisms or in feathery or stellate crystals, hence the term given to this form of stellar phosphates. The earthy phosphates occur as a sediment in the urine when the alkalinity is due to a fixed alkali, or under certain circumstances the deposit may take place within the bladder, and then the phosphates are passed at the end of micturition as a whitish fluid, which is popularly

confounded with spermatorrhœa. The calcium phosphate may be precipitated by heat and produce a cloudiness which may be mistaken for albumen, but is at once dissolved upon making the urine acid. This condition is very frequent in persons suffering from dyspepsia or from debility of any kind. The phosphates may be in great excess, rising in the twenty-four hours to from 7 to 9 grammes (Tessier), whereas the normal amount is not more than 2·5 grammes. And, lastly, the phosphates may be deposited in urine which has undergone decomposition, in which the carbonate of ammonia from the urea combines with the magnesium phosphates, forming the triple salt. This is seen in cystitis, and is due to the introduction of a bacterial ferment.

The clinical significance of an excess of phosphates, to which the term phosphaturia is applied, has been much discussed. It must be remembered that a deposit does not necessarily mean an excess, to determine which a careful analysis of the twenty-four hours' secretion should be made. It has long been thought that there is a relation between the activity of the nerve-tissues and the output of phosphoric acid; but the question cannot yet be considered settled. The amount is increased in wasting diseases, such as phthisis, acute yellow atrophy of the liver, leukæmia, and severe anæmia, whereas it is diminished in acute diseases and during pregnancy.

In a condition termed by Tessier, Ralfe, and others phosphatic diabetes there is polyuria, thirst, emaciation, and a great increase in the excretion of phosphates, which may be as much as from seven to nine grammes in the day. The urine is usually acid, free from sugar, the patients are nervous; in some instances sugar has been present in the urine, and in others it subsequently makes its appearance.

X. Indicanuria.

The substance in the urine which has received this name is the indoxyl-sulphate of potassium, in which form it appears in the urine and is colorless. When concentrated acids or strong oxidizing agents are added to the urine, this substance is decomposed and the indigo set free. It is present only in small quantities in healthy urine. It is derived from the indol, a product formed in the small intestine by the decomposition of the albumen under the influence of bacteria. When absorbed, this is oxidized in the tissues to indoxyl, which combines with the potassium sulphate, forming the above-named substance.

The quantity of indican is increased on a milk diet, in all wasting diseases, and whenever any large quantities of albuminous substances are undergoing rapid decomposition, as in the severer forms of peritonitis and empyema. It is met with also in prolonged constipation and in ileus. Indican has occasionally been found in calculi. Though, as a rule, the urine is colorless when passed, there are instances in which the decompo-

sition has taken place within the body, and a blue color has been noticed immediately after the urine was voided. Sometimes, too, in alkaline urine on exposure there is a bluish film on the surface.

To test for indican, place four or five c. c. of nitric or hydrochloric acid in a test-tube; boil, and add an equal quantity of urine. A bluish ring develops at the point of contact. Add ten c. c. of chloroform and shake the test-tube, and on separation the chloroform has a violet or bluish color due to the presence of indican.

XI. Melanuria.

In melanotic cancer the urine, either at the time of voiding or after exposure to the air, may present a dark color. This pigment is known as melanin, and it may occur in solution or in the form of small granules. The urine may be voided clear, and subsequently, on exposure to the air or on the addition of oxidizing substances, becomes dark. In these cases it contains a chromogen called melanogen which turns dark by oxidation. Von Jaksch has found that "in urine containing melanin or its precursor, melanogen, Prussian blue is formed by adding a nitroprusside, aqueous potash, and an acid. This reaction, however, does not seem to depend on the presence of melanin, as it is not given by that substance when separated from the urine, but apparently by some other at present unknown substance, which is present in traces in normal urine and is increased in cases of melanuria, and also in those conditions where excess of indigo occurs in the urine." (Halliburton.)

XII. Other Substances.

Fat in the urine, or *lipuria*, occurs, according to Halliburton, first, without disease of the kidneys, as in excess of fat in the food, after the administration of cod-liver oil, in fat embolism occurring after fractures, in the fatty degeneration in phosphorus poisoning, in prolonged suppuration as in phthisis and pyæmia, in the lipæmia of diabetes mellitus; secondly, with disease of the kidneys, as in the fatty stage of chronic Bright's disease, in which fat casts are sometimes present, and, according to Ebstein, in pyonephrosis; and, thirdly, in the affection known as chyluria. The urine is usually turbid, but there may be fat drops as well, and fatty crystals have been found.

Lipaciduria is a term applied by von Jaksch to the condition in which there are volatile fatty acids in the urine, such as acetic, butyric, formic, and propionic.

Acetonuria.—Von Jaksch distinguishes the following forms of pathological acetonuria: The febrile, the diabetic, the acetonuria with certain forms of cancer, the form associated with inanition, acetonuria in psychoses, and the acetonuria which results from auto-intoxication. It is doubtful, however, whether the symptoms in these are really due to the

acetone. It may be the substances from which this is formed, particularly the diacetic acid or the oxybutyric acid. The odor of the acetone may be marked in the breath and evident in the urine. Le Nobel's test has been given in the section on diabetes.

Diacetic acid is probably never present in the urine in health. With a solution of ferric chloride it gives a Burgundy-red color. A similar reaction is given by acetic, formic, oxybutyric acids, and it may be present in the urine of patients who are taking antipyrin, thallin, and the salicylates. "If, however, the urine is previously boiled, diacetic acid, if present, still gives the ferric-chloride reaction, but these other substances do not. Fleischer found that the substance which gives the ferric-chloride reaction in diabetic urine is not taken up by ether after the urine has been acidulated with sulphuric acid, whereas ethyl-diacetic acid is soluble in ether." (Halliburton.)

Alcaptonuria.—Aromatic compounds occur in the urine after the administration of carbolic acid or gallic acid, and on exposure to air becomes dark. In carboluria the substance causing the black color is known as hydrochinon. Many years ago Boedeker met with cases in which the urine became dark, owing to the presence of an aromatic compound which he called alcapton. It has been found in cases of consumption, and in other instances in which there are no local lesions or no general disease. The urine may be clear on passing, and then darken on exposure to the air, or on the addition of liquor potassæ. The substance is apparently without clinical significance except in so far as it, with the other aromatic substances, is capable of reducing the Fehling solution, and may be mistaken for sugar.

Choluria and glycosuria have already been considered under jaundice and diabetes.*

IV. URÆMIA.

Under this term is grouped a series of manifestations, chiefly nervous, developing in the course of Bright's disease, and due to the retention within the blood of poisonous materials which should be eliminated in the urine.

Uræmia is usually seen in nephritis, but may occur when the ureters are obstructed, or when the circulation of blood in the kidneys is impeded, as in conditions of extreme engorgement following compression of the renal vessels or in the profound alterations of the blood in cholera.

Two opposite views are held with reference to the production of uræmia: (*a*) That it is due to the accumulation in the blood of excrementitious material—body poisons—which should be thrown off by the

* For further details concerning the urine the student is referred to von Jaksch's Clinical Diagnosis, Tyson on the Urine, and to Halliburton's Text-Book of Chemical Physiology and Pathology.

kidneys. "If, however, from any cause, these organs make default, or if there be any prolonged obstruction to the outflow of urine, accumulation of some or of all the poisons takes place, and the characteristic symptoms are manifested, but the accumulation may be very slow and the earlier symptoms, corresponding to the comparatively small dose of poison, may be very slight; yet they are in kind, though not in degree, as indicative of uræmia as are the more alarming, which appear towards the end, and to which alone the name uræmia is often given." (Carter.) Several poisons having distinct actions have been separated from the urine by Bouchard, two of which produce convulsions, and one of which is narcotic. Bouchard's observations tend strongly to confirm the view now generally held, that the symptoms are caused by the retention of the excretory products. The nature of these poisonous ingredients is not yet known. It was formerly thought that the urea was the offending substance, and it has been found increased in the blood in uræmia. Others hold that it is the accumulation of carbonate of ammonia. It is more probable, however, that there are several toxic agents at work.

(*b*) Traube suggested that the chief symptoms of uræmia, particularly the coma and convulsions, were due to localized œdema of the brain. In favor of this view is the fact that obstruction of the ureters, as by stone, does not necessarily produce uræmia, even if long continued, and in this obstructive suppression neither convulsions nor coma occur. Then, too, uræmia may supervene in a case of chronic Bright's disease in which a large amount of urine is being passed with a fair proportion of solids. Œdema of the brain certainly does occur in some fatal cases—it may be diffuse or localized, but it is not a constant lesion, and cannot explain all the symptoms of uræmia.

Symptoms.—Clinically, acute and chronic uræmia may be recognized, but, for convenience of description, it is perhaps best to follow the division of French writers into *cerebral*, *dyspnœic*, and *gastro-intestinal* forms.

Among the *cerebral* manifestations of uræmia may be described:

(*a*) *Mania.*—This may come on abruptly in an individual who has shown no previous indications of mental trouble, and who may not be known to have Bright's disease. In a remarkable case of this kind which came under my observation the patient became suddenly maniacal and died in six days. More commonly the delirium is less violent, but the patient is noisy, talkative, restless, and sleepless.

(*b*) *Delusional Insanity* (*Folie Brightique*).—Cases are by no means uncommon, and excellent clinical reports have been issued on the subject from several of the asylums of this country, particularly by Bremer, Christian, and Alice Bennett. Delusions of persecution are common. One of my cases committed suicide by jumping out of a window. The condition is of interest medico-legally because of its bearing on testamentary capacity. Profound melancholia may also supervene.

(*c*) *Convulsions.*—These may come on unexpectedly or be preceded by pain in the head and restlessness. The attacks may be general and identical with those of ordinary epilepsy, though the initial cry may not be present. The fits may recur rapidly, and in the interval the patient is usually unconscious. Sometimes the temperature is elevated, but more frequently it is depressed, and may sink rapidly after the attack. Local or Jacksonian epilepsy may occur in most characteristic form in uræmia. A remarkable sequence of the convulsions is blindness—*uræmic amaurosis*—which may persist for several days. This, however, may occur apart from the convulsions. It usually passes off in a day or two. There are no ophthalmoscopic changes. Sometimes uræmic deafness supervenes, and is probably also a cerebral manifestation. It may also occur in connection with persistent headache, nausea, and other gastric symptoms.

(*d*) *Coma.*—Unconsciousness invariably accompanies the general convulsions, but a coma may develop gradually without any convulsive seizures. Frequently it is preceded by headache, and the patient gradually becomes dull and apathetic. In these cases there may have been no previous indications of renal disease, and unless the urine is examined the nature of the case may be overlooked. Twitchings of the muscles occur, particularly in the face and hands, but there are many cases of coma in which the muscles are not involved. In some of these cases a condition of torpor persists for weeks or even months. The tongue is usually furred and the breath very foul and heavy.

(*e*) *Local Palsies.*—In the course of chronic Bright's disease hemiplegia or monoplegia may come on spontaneously or follow a convulsion, and post mortem no gross lesions of the brain be found, but only a localized or diffused œdema. These cases, which are not very uncommon, may simulate almost every form of organic paralysis of cerebral origin.

(*f*) Of other cerebral symptoms, headache is important. It is most often occipital and extends to the neck. It may be an early feature and associated with giddiness. Other nervous symptoms of uræmia are intense itching of the skin, numbness and tingling in the fingers, and cramps in the muscles of the calves, particularly at night.

Uræmic dyspnœa is classified by Palmer Howard as follows: (1) Continuous dyspnœa; (2) paroxysmal dyspnœa; (3) both types alternating; and (4) Cheyne-Stokes breathing. The attacks of dyspnœa are most commonly nocturnal; the patient may sit up, gasp for breath, and evince as much distress as in true asthma. Occasionally the breathing is noisy and stridulous. The Cheyne-Stokes type may persist for weeks, and is not necessarily associated with coma. I have seen it in a man who travelled over a hundred miles to consult a physician. In another instance a patient, up and about, could only when at meals feed himself in the apnœa period. Though usually of serious omen and occurring with coma and

other symptoms, recovery may follow even after persistence for weeks or even months.

The *gastro-intestinal* manifestations of uræmia often set in with abruptness. Uncontrollable vomiting may come on and its cause be quite unrecognizable. A young married woman was admitted to my wards in the Montreal General Hospital with persistent vomiting of four or five days' duration. The urine was slightly albuminous, but she had none of the usual signs of uræmia, and the case was not regarded as one of Bright's disease. The vomiting persisted and caused death. The post-mortem showed extensive sclerosis of both kidneys. The attacks may be preceded by nausea and may be associated with diarrhœa. In some instances the diarrhœa may come on without the vomiting; sometimes it is profuse and associated with an intense catarrhal or even diphtheritic inflammation of the colon.

A special uræmic stomatitis has been described (Barie) in which the mucosa of the lips, gums, and tongue is swollen and erythematous. The saliva may be increased, and there is difficulty in swallowing and in mastication. The tongue is usually very foul and the breath heavy and fetid.

Diagnosis.—Uræmia may be confounded with:

(*a*) Cerebral lesions, such as hæmorrhage, meningitis, or even tumor. In apoplexy, which is so commonly associated with kidney disease and stiff arteries, the sudden loss of consciousness, particularly if with convulsions, may simulate a uræmic attack; but the mode of onset, the existence of complete hemiplegia, with conjugate deviation of the eyes, suggest hæmorrhage. As already noted, there are cases of uræmic hemiplegia or monoplegia which cannot be separated from those of organic lesion and which post mortem show no trace of coarse disease of the brain. I know of an instance in which a consultation was held upon the propriety of operation in a case of hemiplegia believed to be due to subdural hæmorrhage which post mortem was shown to be uræmic. Indeed, in some of these cases it is quite impossible to distinguish between the two conditions. So, too, cases of meningitis, in a condition of deep coma, with perhaps slight fever, furred tongue, and without localizing symptoms, may readily be confounded with uræmia.

(*b*) With certain infectious diseases. Uræmia may persist for weeks or months and the patient lies in a condition of torpor or even unconsciousness, with a heavily coated, perhaps dry, tongue, muscular twitchings, a rapid feeble pulse, with slight fever. This state not unnaturally suggests the existence of one of the infectious diseases. Cases of the kind are not uncommon, and I have known them to be mistaken for typhoid fever and for miliary tuberculosis.

(*c*) Uræmic coma may be confounded with poisoning by alcohol or opium. In opium poisoning the pupils are contracted; in alcoholism they are more commonly dilated. In uræmia they are not constant; they may be either widely dilated or of medium size. The examination of the eye

ground should be made to determine the presence or absence of albuminuric retinitis. The urine should be drawn off and examined. The odor of the breath sometimes gives an important hint.

The condition of the heart and arteries should also be taken into account. Sudden uræmic coma is more common in the chronic interstitial nephritis. The character of the delirium in alcoholism is sometimes important, and the coma is not so deep as in uræmia or opium poisoning. It may for a time be impossible to determine whether the condition is due to uræmia, profound alcoholism, or hæmorrhage into the pons Varolii. The treatment will be considered under chronic Bright's disease.

V. ACUTE BRIGHT'S DISEASE.

Definition.—Acute diffuse nephritis, due to the action of cold or of toxic agents upon the kidneys.

In all instances changes exist in the epithelial, vascular, and intertubular tissues, which vary in intensity in different forms; hence writers have described a tubular, a glomerular, and an acute interstitial nephritis. Delafield recognizes *acute exudative* and *acute productive* forms, the latter characterized by proliferation of the connective-tissue stroma and of the cells of the Malpighian tufts.

Etiology.—The following are the principal causes of acute nephritis:

(1) Cold. Exposure to cold and wet is one of the most common causes. It is particularly prone to follow exposure after a drinking-bout.

(2) The poisons of the specific fevers, particularly scarlet fever, less commonly typhoid fever, measles, diphtheria, small-pox, chicken-pox, cholera, yellow fever, meningitis, and, very rarely, dysentery. Acute nephritis is not often associated with syphilis. In acute tuberculosis nephritis is not uncommon. It may also occur in septicæmia.

(3) Toxic agents, such as turpentine, cantharides, chlorate of potash, and carbolic acid may cause an acute congestion which sometimes terminates in nephritis. Alcohol probably never excites an acute nephritis.

(4) Pregnancy, in which the condition is thought by some to result from compression of the renal veins, although this is not yet finally settled. The condition may in reality be due to toxic products as yet undetermined.

(5) Acute nephritis occurs occasionally in connection with extensive lesions of the skin, as in burns or in chronic skin-diseases.

Morbid Anatomy.—The kidneys may present to the naked eye in mild cases no evident alterations. When seen early in more severe forms the organs are congested, swollen, dark, and the section may drip blood. In other instances the surface is pale and mottled, the capsule strips off readily, and the cortex is swollen, turbid, and of a grayish-red color, while the pyramids have an intense beefy-red tint. The glomeruli in some in-

stances stand out plainly, deeply swollen and congested; in other instances they are pale.

The histology may be thus summarized: (*a*) Glomerular changes. In a majority of the cases of nephritis due to toxic agents, which reach the kidney through the blood-vessels, the tufts suffer first, and there is either an acute intracapillary glomerulitis, in which the capillaries become filled with cells and thrombi or involvement of the epithelium of the tuft and of Bowman's capsule, the cavity of which contains leucocytes and red blood-corpuscles. Hyaline degeneration of the contents and of the walls of the capillaries of the tufts is an extremely common event. These processes are perhaps best marked in scarlatinal nephritis. There may be proliferation about Bowman's capsule. These changes interfere with the circulation in the tufts and seriously influence the nutrition of the tubular structures beyond them.

(*b*) The alterations in the tubular epithelium consist in cloudy swelling, fatty change, and hyaline degeneration. In the convoluted tubules, the accumulation of altered cells with leucocytes and blood-corpuscles causes the enlargement and swelling of the organ. The epithelial cells lose their striation, their nuclei are obscured, and hyaline droplets often accumulate in them.

(*c*) Interstitial changes. In the milder forms a simple inflammatory exudate—serum mixed with leucocytes and red blood-corpuscles—exists between the tubules. In severer cases areas of small-celled infiltration occur about the capsules and between the convoluted tubes. These changes may be wide-spread and uniform throughout the organs or more intense in certain regions.

Symptoms.—The onset is usually sudden, and when the nephritis follows cold, dropsy may be noticed within twenty-four hours. After fevers the onset is less abrupt, but the patient gradually becomes pale and a puffiness of the face or swelling of the ankles is first noticed. In children there may at the outset be convulsions. Chilliness or rigors initiate the attack in a limited number of cases. Pain in the back, nausea, and vomiting may be present. The fever is variable. Many cases in adults have no rise in temperature. In young children with nephritis from cold or scarlet fever the temperature may, for a few days, range from 101° to 103°.

The most characteristic symptoms are the urinary changes. There may at first be suppression, more commonly the urine is scanty and highly colored and contains blood, albumen, and tube-casts. The quantity is reduced and only four or five ounces may be passed in the twenty-four hours; the specific gravity is high—1·025, or even more; the color varies from a smoky to a deep porter color, but is seldom bright red. On standing there is a heavy deposit; microscopically there are blood-corpuscles, epithelium from the urinary passages, and casts, hyaline, blood, and epithelial in character. The albumen is abundant, forming a curdy, thick pre-

cipitate. The total excretion of urea is reduced, though the percentage is high.

Anæmia is an early and marked symptom. In cases of extensive dropsy, effusion may take place into the pleuræ and peritoænum. There are cases of scarlatinal nephritis in which the dropsy of the extremities is trivial and effusion into the pleuræ extensive. The lungs may become œdematous. In rare cases there is œdema of the glottis. Epistaxis may occur or cutaneous ecchymoses may develop in the course of the disease.

The pulse may be hard, the tension increased, and the second aortic sound accentuated. Occasionally dilatation of the heart comes on rapidly and may cause sudden death (Goodhart). The skin is dry and it may be difficult to induce sweating.

Uræmic symptoms develop in a limited number of cases. They may occur at the onset with suppression, more commonly later in the disease. Ocular changes are not so common in acute as in chronic Bright's disease, but hæmorrhagic retinitis may occur and occasionally papillitis.

The course of acute Bright's disease varies considerably. The description just given is of the form which most commonly follows cold or scarlet fever. In many of the febrile cases dropsy is not a prominent symptom, and the diagnosis rests rather with the examination of the urine. Moreover, the condition may be transient and less serious. In other cases, as in the acute nephritis of typhoid fever, there may be hæmaturia and pronounced signs of interference with the renal function. The most intense acute nephritis may exist without anasarca.

In scarlatinal nephritis, in which the glomeruli are most seriously affected, suppression of the urine may be an early symptom, the dropsy is apt to be extreme, and uræmic manifestations are common. Acute Bright's disease in children, however, may set in very insidiously and be associated with transient or slight œdema, and the symptoms may point rather to affection of the digestive system or to brain-disease.

Diagnosis.—It is very important to bear in mind that the most serious involvement of the kidneys may be manifested only by slight œdema of the feet or puffiness of the eyelids, without impairment of the general health. The first indication of trouble may be a uræmic convulsion. This is particularly the case in the acute nephritis of pregnancy, and it is a good rule for the practitioner, when engaged to attend a case, invariably to ask that during the seventh and eighth months the urine should occasionally be sent for examination.

In nephritis from cold and in scarlet fever the symptoms are usually marked and the diagnosis is rarely in doubt. As already mentioned, every case in which albumen is present must not be called acute Bright's disease, not even if tube-casts be present. Thus the common febrile albuminuria, although it represents the first link in the chain of events leading to acute Bright's disease, should not be placed in the same category.

There are occasional cases of acute Bright's disease with anasarca, in

which albumen is either absent or present only as a trace. This is a rare condition. Tube-casts are usually found, and the absence of albumen is rarely permanent. The urine may be reduced in amount.

The character of the casts is of use in the diagnosis of the form of Bright's disease, but scarcely of such extreme value as has been stated. Thus, the hyaline and granular casts are common to all varieties. The blood and epithelial casts, particularly those made up of leucocytes, are most common in the acute cases.

Prognosis.—The outlook varies somewhat with the cause of the disease. Recoveries in the form following exposure to cold are much more frequent than after scarlatinal nephritis. In young children the mortality is high, amounting to at least one third of the cases. Serious symptoms are low arterial tension, the occurrence of uræmia, and effusion into the serous sacs. The persistence of the dropsy after the first month, intense pallor, and a large amount of albumen indicate the possibility of the disease becoming chronic. For some months after the disappearance of the dropsy there may be traces of albumen and a few tube-casts.

In a week or ten days, in a case of scarlatinal nephritis, if the progress is favorable, the dropsy diminishes, the urine increases, the albumen lessens, and by the end of a month the dropsy has disappeared and the urine is nearly free. In very young children the course may be rapid, and I have known the urine to be free from albumen in the fourth week. Other cases are more insidious, and though the dropsy may disappear, the albumen persists in the urine, the anæmia is marked, and the condition becomes chronic or, after several recurrences of the dropsy, improves and complete recovery takes place.

Treatment.—The patient should be in bed and there remain until all traces of the disease have disappeared. As sweating plays such an important part in the treatment, it is well, if possible, to accustom the patient to blankets. He should also be clad in thin Canton flannel.

The diet should consist of milk or butter-milk, gruels made of arrow-root or oat-meal, barley water, and, if necessary, beef tea and chicken broth. It is better, if possible, to confine the patient to a strictly milk diet. As convalescence is established, bread and butter, lettuce, water-cress, grapes, oranges, and other fruits may be given. The return to a meat diet should be gradual.

The patient should drink freely of alkaline mineral waters, ordinary water, or lemonade. The fluids keep the kidneys flushed and wash out the *débris* from the tubes. A useful drink is a drachm of cream of tartar in a pint of boiling water, to which may be added the juice of half a lemon and a little sugar. Taken when cold, this is a pleasant and satisfactory diluent drink.

No remedies, so far as known, control directly the changes which are going on in the kidneys. The indications are: (1) To give the excretory

function of the kidney rest by utilizing the skin and the bowels, in the hope that the natural processes may be sufficient to effect a cure; (2) to meet the symptoms as they arise.

In a case of scarlet fever it may occasionally be possible to avert an attack, the premonitory symptoms of which are marked increase in the arterial tension and the presence of blood coloring matter in the urine (Mahomed). An active saline cathartic may completely relieve this condition.

At the onset, when there is pain in the back or hæmaturia, the dry or wet cups give relief. The latter should not be used in children. Warm poultices are often grateful. In cases which set in with suppression of urine, these measures should be adopted, and in addition the hot bath with subsequent pack, copious diluents, and a free purge. The dropsy is best treated by hydrotherapy—either the hot bath, the wet pack, or the hot-air bath. In children the wet pack is usually satisfactory. It is applied by wringing a blanket out of hot water, wrapping the child in it, covering this with a dry blanket, and then with a rubber cloth. In this the child may remain for an hour. It may be repeated daily. In the case of adults, the hot-air bath or the vapor bath may be conveniently given by allowing the vapor or air to pass from a funnel beneath the bed-clothes, which are raised on a low cradle. More efficient, as a rule, is a hot bath of from fifteen or twenty minutes, after which the patient is wrapped in blankets. The sweating produced by these measures is usually profuse, rarely exhausting, and in a majority of cases the dropsy can in this way be relieved. There are some cases, however, in which the skin does not respond to the baths, and if the symptoms are serious, particularly if uræmia supervenes, jaborandi or its active principle, pilocarpine, may be used. The latter may be given hypodermically, in doses of from a sixth to an eighth of a grain in adults, and from a twentieth to a twelfth of a grain in children from two to ten years. It is a drug to be used with care. I abandoned its employment for many years, after having several cases of serious collapse. Latterly I have resumed its use, often with benefit.

The bowels should be kept open by a morning saline purge; in children the fluid magnesia is readily taken; in adults the sulphate of magnesia may be given by Hay's method, in concentrated form, in the morning, before anything is taken into the stomach. In Bright's disease it not infrequently causes vomiting. The compound powder of jalap, in half-drachm doses, or, if necessary, elaterium may be used. If the dropsy is not extreme, the urine not very concentrated, and uræmic symptoms are not present, the bowels should be kept loose without active purgation. If these measures fail to reduce the dropsy and it has become extreme, the skin may be punctured with a lancet or drained by a small silver canula (Southey's tube), which is inserted beneath it. A fine aspirator needle may be used, and the fluid allowed to drain through a piece of long, narrow rubber tubing into a vessel beneath the bed. If the dyspnœa is marked,

owing to pressure of fluid in the pleuræ, aspiration should be performed. In rare instances the ascites is extreme and may require paracentesis, or a Southey's tube may be inserted and the fluid gradually withdrawn. If uræmic convulsions occur, the intensity of the paroxysms may be limited by the use of chloroform; to an adult a pilocarpine injection should be at once given, and from a robust, strong man twenty ounces of blood may be withdrawn. In children the loins may be dry cupped, the wet pack used, and a brisk purgative given. Bromide of potassium and chloral sometimes prove useful.

Vomiting may be relieved by ice and by restricting the amount of food. Drop doses of creosote, iodine, and carbolic acid may be given. The dilute hydrocyanic acid with bismuth is often effectual.

The question of the use of diuretics in acute Bright's disease is not yet settled. The best diuretic, after all, is water, which may be taken freely with the citrate of potash or the benzoate of soda, salts which are held to favor the conversion of the urates into less irritating and more easily excreted compounds. Digitalis and strophanthus are useful diuretics, and may be employed without risk when the arterial tension is low and the cardiac impulse is not forcible. I have never seen any injurious effects from their employment after the early symptoms had lessened in intensity.

For the persistent albuminuria, I agree with Roberts and Rosenstein that we have no remedy of the slightest value. Nothing indicates more clearly our helplessness in controlling kidney metabolism than inability to meet this common symptom. Astringents, alkalies, nitroglycerin, and mercury have been recommended.

For the anæmia always associated with acute Bright's disease iron should be employed. It should not be given until the acute symptoms have subsided. In the adult it may be used in the form of the perchloride in increasing doses, as convalescence proceeds. In children, the syrup of the iodide of iron or the syrup of the phosphate of iron are better preparations. The dilatation of the heart is best treated with digitalis, strophanthus, and strychnia.

In the convalescence from acute Bright's disease, care should be taken to guard the patient against cold. The diet should still consist chiefly of milk and a return to mixed food should be gradual. A change of air is often beneficial, particularly a residence in a warm, equable climate.

VI. CHRONIC BRIGHT'S DISEASE.

Here, too, in all forms we deal with a diffuse process, involving epithelial, interstitial, and glomerular tissues. Clinically two groups are recognized—(*a*) the chronic parenchymatous nephritis, which follows the acute attack or comes on insidiously, is characterized by marked dropsy, and post mortem by the *large white kidney*. In the later stages of this

process the kidney may be smaller—a condition known as the *small white kidney*; (*b*) chronic interstitial nephritis, in which dropsy is not common and the cardio-vascular changes are pronounced. Delafield recognizes a chronic diffuse nephritis with exudation and a chronic productive diffuse nephritis without exudation, the latter corresponding to the contracted kidney of authors.

The amyloid kidney is usually spoken of as a variety of Bright's disease, but in reality it is a degeneration which may accompany any form of nephritis.

Chronic Parenchymatous Nephritis

(Chronic Desquamative and Chronic Tubal Nephritis; Chronic Diffuse Nephritis with Exudation).

Etiology.—In many cases the disease follows the acute nephritis of cold, scarlet fever, or pregnancy. More frequently than is usually stated the disease has an insidious onset and occurs independently of any acute attack. The fevers may play an important *rôle* in certain of these cases. Rosenstein, Bartels, and, in this country, I. E. Atkinson have laid special stress upon malaria as a cause. No instance of the kind has fallen under my observation during the past seven years, in which time several hundred cases of malaria have been under my treatment. Beer and alcohol are believed to lead to this form of nephritis. In chronic suppuration, syphilis, and tuberculosis the diffuse parenchymatous nephritis is not uncommon, and is usually associated with amyloid disease. Males are rather more subject to the affection than females. It is met with most commonly in young adults, and is by no means infrequent in children as a sequence of scarlatinal nephritis.

Morbid Anatomy.—Several varieties of this form have been recognized. The most common is the *large white kidney* of Wilks, in which the organ is enlarged, the capsule is thin, and the surface white with the stellate veins injected. On section the cortex is swollen and yellowish white in color, and often presents opaque areas. The pyramids may be deeply congested. On microscopical examination it is seen that the epithelium is granular and fatty, and the tubules of the cortex are distended, and contain tube-casts. Hyaline changes are also present in the epithelial cells. The glomeruli are large, the capsules thickened, the capillaries show hyaline changes, and the epithelium of the tuft and of the capsule is extensively altered. The interstitial tissue is everywhere increased, though not to an extreme degree.

The second variety of this form results from the gradual increase in the connective tissue and the subsequent shrinkage, forming what is called the *small white kidney* or the pale granular kidney. It is doubtful whether this is always preceded by the large white kidney. Some observers hold that it may be a primary independent form. The capsule is thick-

ened and the surface is rough and granular. On section the resistance is greatly increased, the cortex is reduced and presents numerous opaque white or whitish-yellow foci, consisting of accumulations of fatty epithelium in the convoluted tubules. This combination of contracted kidney with the areas of marked fatty degeneration has given the name of small granular, fatty kidney to this form. The interstitial changes are marked, many of the glomeruli are destroyed, the degeneration of epithelium in the convoluted tubules is wide-spread, and the arteries are greatly thickened.

Belonging to this chronic tubal nephritis is a variety known as the *chronic hæmorrhagic nephritis*, in which the organs are enlarged, yellowish white in color, and in the cortex are many brownish-red areas, due to hæmorrhage into and about the tubes. In other respects the changes are identical with those in the large white kidney.

Of changes in the other organs the most marked are thickening of the blood-vessels and hypertrophy of the left heart.

Symptoms.—Following an acute nephritis, the disease may present, in a modified way, the symptoms of that affection. In many cases it sets in insidiously, and after an attack of dyspepsia or a period of failing health and loss of strength the patient becomes pale and puffiness of the eyelids or swollen feet are noticed in the morning.

The symptoms are as follows: The urine is, as a rule, diminished in quantity, often scanty. It has a dirty-yellow, sometimes smoky, color and is turbid from the presence of urates. On standing, a heavy sediment falls, in which are found numerous tube-casts of various forms and sizes, hyaline, both large and small, epithelial, granular, and fatty casts. Leucocytes are abundant; red blood-corpuscles are frequently met with, and epithelium from the kidneys and pelves. The albumen is abundant and may amount to one half or one third of the urine boiled. It is more abundant in the urine passed during the day. The specific gravity may be high in the early stages—from 1·020 to 1·025—though in the later stages it is lower. The urea is always reduced in quantity.

Dropsy is a marked and obstinate symptom of this form of Bright's disease. The face is pale and puffy, and in the morning the eyelids are œdematous. The anasarca is general, and there may be involvement of the serous sacs. In these chronic cases associated with large white kidney there is often a distinctive appearance in the face; the complexion is pasty, the pallor marked, and the eyelids are œdematous. The dropsy is peculiarly obstinate. Uræmic symptoms are common, though convulsions are perhaps less frequent than in the interstitial nephritis.

The tension of the pulse is usually increased; the vessels ultimately become stiff and the heart hypertrophied, though there are instances of this form of nephritis in which the heart is not enlarged. The aortic second sound is accentuated. Retinal changes though less frequent than in the chronic interstitial nephritis, occur in a considerable number of cases.

Gastro-intestinal symptoms are common. Vomiting is frequently a distressing and serious symptom, and diarrhœa may be profuse. Ulceration of the colon may occur and prove fatal.

It is sometimes impossible to determine, even by the most careful examination of the urine or by analysis of the symptoms, whether the condition of the kidney is that of the large white or of the small white form. In cases, however, which have lasted for several years, with the progressive increase in the renal connective tissue and the cardio-vascular changes, the clinical picture may approach, in certain respects, that of the contracted kidney. The urine is increased, with low specific gravity. It is often turbid, may contain traces of blood, the tube-casts are numerous and of every variety of form and size, and the albumen is abundant. Dropsy is usually present, though not so extensive as in the early stages.

The *prognosis* is extremely grave. In a case which has persisted for more than a year recovery rarely takes place. Death is caused either by great effusion with œdema of the lungs, by uræmia, or by secondary inflammation of the serous membranes. Occasionally in children, even when the disease has persisted for two years, the symptoms disappear and recovery takes place.

Treatment.—Essentially the same treatment should be carried out as in acute Bright's disease. Milk or butter-milk should constitute the chief article of food. The dropsy should be treated by hydrotherapy. Iron preparations should be given freely. The acetate of potash and digitalis are useful in increasing the flow of urine. Basham's mixture given in plenty of water will be found beneficial.

Chronic Interstitial Nephritis.

(Contracted Kidney; Granular Kidney; Cirrhosis of the Kidney; Gouty Kidney; Renal Sclerosis).

Sclerosis of the kidney is met with (*a*) as an occasional sequence of the large white kidney, forming the so-called pale granular or secondary contracted kidney; (*b*) as an independent primary affection; (*c*) as a sequence of arterio-sclerosis.

Etiology.—The primary form is chronic from the outset, and is a slow, creeping degeneration of the kidney substance—in many respects only an anticipation of the gradual changes which take place in the organ in extreme old age. In many cases no satisfactory cause can be assigned. In others there are hereditary influences, as in the remarkable family studied by Dickenson, in which a pronounced tendency to chronic Bright's disease occurred in four generations. Families in which the arteries tend to degenerate early are more prone to interstitial nephritis. Syphilis is held by some to be a cause. Alcohol probably plays an important part, particularly in conjunction with other factors. Dietetic influences are at work in many cases. Some believe excessive use of meat is injurious, since it

increases the materials out of which uric acid is formed. By many a functional disorder of the liver, leading to lithæmia, is regarded as the most efficient factor. It is quite possible that in persons who habitually eat and drink too much the work thrown upon this organ is excessive, and the elaboration of certain materials so defective that in their excretion from the general circulation they irritate the kidneys.

Actual gout, which in England is a common cause of interstitial nephritis, is not an important factor here. On the other hand, the nutritional disorder known as lithæmia is very common, either with or without dyspepsia. Lead, as is well known, may produce renal sclerosis, but it is a minor factor in comparison with other causes. It is doubtful if climate has any influence. Purdy regards the cold, moist regions of the Northeastern States as specially favorable to the disease.

Among factors which may account for the prevalence of chronic Bright's disease in the better classes in this country may be mentioned the intense worry and strain of business, combined, as they often are, with habits of hurried and over eating and a lack of proper exercise. Males are more commonly attacked than females. Under twenty-five years of age it is a rare disease; between twenty-five and forty a few well-marked cases occur; between forty and sixty it is common.

Morbid Anatomy.—The kidneys are usually small, and together may weigh no more than an ounce and a half. The capsule is thick and adherent; the surface of the organ irregular and covered with small nodules, which have given to it the name of granular kidney. In stripping off the capsule, portions of the kidney substance are removed. Small cysts are frequently seen on the surface. The color is usually reddish, often a very dark red. On section the substance is tough and resists cutting; the cortex is thin and may measure no more than a couple of millimetres. The pyramids are less wasted. The small arteries are greatly thickened and stand out prominently. The fat about the pelvis is greatly increased.

Microscopically there is seen a marked increase in the connective tissue and degeneration and atrophy of the secreting structures, glomerular and tubal, the former being most predominant and giving the main characters to the lesion. The following are the most important changes:

(*a*) An increase in the fibrous elements, widely distributed throughout the organ, but more advanced in the cortex, particularly in the tissue between the medullary rays. In the pyramids the distribution of new growth is less patchy and more diffuse. In the early stages of the process there is a small-celled infiltration between the tubes and around the glomeruli, and finally this becomes fibrillated and is seen encircling the tubules and Bowman's capsules, around the latter often forming concentric layers.

(*b*) The changes in the glomeruli are striking, and in advanced cases a very considerable number of them have undergone complete atrophy and are represented as densely encapsulated hyaline structures. The atrophy is partly due to changes in the capillary walls and multiplication of cells

between the loops, partly to extensive hyaline degeneration, and in part, no doubt, to the alterations in the afferent vessels. The normal glomeruli usually show some thickening of the capsule and increase in the cells of the tufts.

(*c*) The tubules show changes in the epithelium, which vary a good deal in different localities. Where the connective-tissue growth is most advanced they are greatly atrophied and the epithelium may be represented by small cubical cells. In other instances the epithelium has entirely disappeared. On the other hand, in the regions represented by the projecting granules the tubules are usually dilated, and the epithelium shows hyaline, fatty, and granular changes. Very many of them contain dark masses of epithelial *débris* and tube-casts. In the interstitial tissue and in the tubules there may be pigmentary changes due to hæmorrhage. The dilatation of the tubules may reach an extreme grade, forming definite cysts.

(*d*) The arteries show an advanced sclerosis. The intima is greatly thickened and there are changes in the adventitia and in the media, consisting in increase in the thickness due to proliferation of the connective tissue, in the latter coat at the expense of the muscular elements.

The view most generally entertained at present is that the essential lesion is in the secreting tissues of the tubules and the glomeruli, and that the connective-tissue overgrowth is secondary to this. Greenfield holds that the primary change is in most instances in the glomeruli, to which both the degeneration in the epithelium of the convoluted tubules and the increase in the intertubular connective tissue are secondary.

Associated with contracted kidney are general arterio-sclerosis and hypertrophy of the heart. The changes in the arteries have already been described in the section on arterio-sclerosis. The hypertrophy of the heart is almost constant. I do not remember ever to have seen a well-marked instance of contracted kidney without some hypertrophy of the left ventricle, and the enlargement may reach an extreme grade. The variations depend, no doubt, in part upon the extent of the diffuse arterial degeneration, and there are instances in which the term *cor bovinum* may be applied to the enlarged organ. In such cases the hypertrophy is not confined to the left ventricle, but involves the entire heart. The explanation of this hypertrophy has been much discussed. It was at first held to be due to the increased work thrown upon the organ in driving the impure blood through the capillary system. Basing his opinion upon the supposed muscular increase in the smaller arteries, Johnson regarded the hypertrophy as effort to overcome a sort of stop-cock action of these vessels, which, under the influence of the irritating ingredient in the blood, contracted and increased greatly the peripheral resistance. Traube believed that the obliteration of a large number of capillary territories in the kidney materially raised the arterial pressure, and in this way led to the hypertrophy of the heart; an additional factor, he thought, was the

diminished excretion of water, which also heightened the pressure within the blood-vessels.

In our present knowledge the most satisfactory explanation is that given by Cohnheim, which is thus clearly and succinctly put by Fagge: "He gives reasons for thinking that the activity of the circulation through the kidneys at any moment—in other words, the state of the smaller renal arteries as regards contraction or dilatation—depends not (as in the case of the tissues generally) upon the need of those organs for blood, but solely upon the amount of material for the urinary secretion that the circulatory fluid happens then to contain. This suggestion has bearings . . . upon the development of hypertrophy in one kidney when the other has been entirely destroyed. But another consequence deducible from it is that when parts of both kidneys have undergone atrophy, the blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact. But in order that such a quantity of blood should pass through the restricted capillary area now open to it, an excessive pressure must obviously be necessary. This can be brought to bear only by the exertion of more than the normal degree of force on the part of the left ventricle, combined with the maintenance of a corresponding resistance in all other districts of the arterial system. And so one can account at once for the high arterial pressure and for the cardio-vascular changes that are secondary to it."

Symptoms.—Perhaps a majority of the cases are latent, and are not recognized until the occurrence of one of the serious or fatal complications. Even an advanced grade of contracted kidney may be compatible with great mental and bodily activity. There may have been no symptoms whatever to suggest to the patient the existence of a serious malady. In other cases the general health is disturbed. The patient complains of lassitude, is sleepless, has to get up at night to micturate; the digestion is disordered, the tongue is furred; there are complaints of headache, failing vision, and breathlessness on exertion.

So complex and varied is the clinical picture of chronic Bright's disease that it will be best to consider the symptoms under the various systems.

Urinary System.—The amount of urine is usually increased, and from two to four litres may be passed. Frequently the patient has to get up two or three times during the night to empty the bladder, and there is increased thirst. It is for these symptoms occasionally that relief is sought. It is to be remembered, however, that frequent micturition at night may be associated with irritability of the prostate and, in certain cases, with hyperacidity of the urine. The secretion is clear, the mucus cloud is well marked, but there is no definite sediment. The color is a light yellow, and the specific gravity ranges from 1·005 to 1·012. Traces of albumen are found, but may be absent at times, particularly in the early morning urine. It is often simply a slight cloudiness, and may be apparent only

with the more delicate tests. The sediment is scanty, and in it a few hyaline or granular casts are found. The quantity of the solid constituents of the urine is, as a rule, diminished, though in some instances the urea may be excreted in full amount. In attacks of dyspepsia or bronchitis, or in the later stages when the heart fails, the quantity of albumen may be greatly increased and the urine diminished. Occasionally blood occurs in the urine, and there may even be hæmaturia (S. West).

Circulatory System.—The pulse is hard, the tension increased, and the vessel wall, as a rule, thickened. As already mentioned, a distinction must be made between increased tension and thickening of the arterial wall. The tension may be plus in a normal vessel, but in chronic Bright's disease it is more common to find the tension is increased and the artery stiff.

A pulse of increased tension has the following characters: (*a*) It is hard and incompressible, requiring a good deal of force to overcome it; (*b*) it may be impossible to obliterate the pulse wave by any pressure on the vessel; (*c*) it is persistent, and in the intervals between the beats the vessel feels full and can be rolled beneath the finger. These characters may be present in a vessel the walls of which are little, if at all, increased in thickness. To estimate the latter the pulse wave should be obliterated in the radial, and the vessel wall felt beyond it. In a perfectly normal vessel the arterial coats, under these circumstances, cannot be differentiated from the surrounding tissue; whereas, if thickened, the vessel can be rolled beneath the finger. Persistent high tension is one of the earliest and most important symptoms of interstitial nephritis. The cardiac features are equally important, though often less obvious. Hypertrophy of the left ventricle occurs to overcome the resistance offered in the arteries. The enlargement of the heart ultimately becomes more general. The apex is displaced downward and to the left; the impulse is forcible and may be heaving. In elderly persons with emphysema, the displacement of the apex may not be evident. The first sound at the apex may be duplicated; more commonly the second sound at the aortic cartilage is accentuated, a very characteristic sign of increased tension. The sound in extreme cases may have a bell-like quality. In many cases a systolic murmur develops at the apex, probably as a result of relative insufficiency. It may be loud and transmitted to the axilla. Finally the hypertrophy fails, the heart becomes dilated, gallop rhythm is present, and the general condition is that of a chronic heart-lesion.

Respiratory System.—Sudden œdema of the glottis may occur. Effusion into the pleuræ or sudden œdema of the lungs may prove fatal. Acute pleurisy and pneumonia are not uncommon. Bronchitis is a frequent accompaniment, particularly in the winter. Sudden attacks of oppressed breathing, particularly at night, are not infrequent. This is often a uræmic symptom, but is sometimes cardiac. The patient may sit up in bed and gasp for breath, as in true asthma. Cheyne-Stokes breath-

ing may be present, most commonly toward the close, but the patient may be walking about and even attending to his occupation.

Digestive System.—Dyspepsia and loss of appetite are common. Severe and uncontrollable vomiting may be the first symptom. This is usually regarded as a manifestation of uræmia, but it may be present without any other indications, and I have known it to prove fatal without any suspicion that chronic Bright's disease was present. Severe and even fatal diarrhœa may develop. The tongue may be coated and the breath heavy and urinous.

Nervous System.—Various cerebral manifestations have already been mentioned under uræmia, and they are among the most important of the features of chronic Bright's disease. Cerebral apoplexy is closely related to interstitial nephritis. The hæmorrhage may take place into the meninges or the cerebrum. It is usually associated with marked changes in the vessels. Neuralgias, in various regions, are not uncommon.

Special Senses.—Troubles in vision may be the first symptom of the disease. It is remarkable in how many cases of interstitial nephritis the condition is diagnosed first by the ophthalmic surgeon. The flame-shaped hæmorrhages are the most common. Less frequent is diffuse retinitis or papillitis. Sudden blindness may supervene without retinal changes—uræmic amaurosis. Auditory troubles are by no means infrequent in chronic Bright's disease. Ringing in the ears, with dizziness, is not uncommon. Various forms of deafness may occur.

Skin.—Œdema is not common in interstitial nephritis. Slight puffiness of the ankles may be present, but in a majority of the cases dropsy does not supervene. When extensive, it is almost always the result of gradual failure of the hypertrophied heart. The skin is often dry and pale, and sweats are not common. In some instances the sweat may deposit a white frost of urea on the surface of the skin. Eczema is a common accompaniment of chronic interstitial nephritis. Tingling of the fingers or numbness and pallor—the dead fingers—are not, as some suppose, in any way peculiar to Bright's disease. Intolerable itching of the skin may be present, and cramps in the muscles are by no means rare.

Hæmorrhages are not infrequent; thus, epistaxis may occur and prove serious. Purpura may develop. Broncho-pulmonary hæmorrhages are said, by some French writers, to be common, but no instance of it has come under my observation. Ascites is rare except in association with cirrhosis of the liver.

Diagnosis.—The autopsy often discloses the true nature of the disease, one of the many intercurrent affections of which may have proved fatal. The early stages of interstitial nephritis are not recognizable. In a patient with increased pulse tension (particularly if the vessel wall is sclerotic), with the apex beat of the heart dislocated to the left, the second aortic sound ringing and accentuated, the urine abundant and of low specific gravity, with a trace of albumen and an occasional hyaline or

granular cast, the diagnosis of interstitial nephritis may be safely made. Of all the indications, that offered by the pulse is the most important Persistent high tension with thickening of the arterial wall in a man under fifty means that serious mischief has already taken place, that cardio-vascular changes are certainly, and renal most probably, present. It is important in the diagnosis of this condition not to rest content with a single examination of the urine. Both the evening and the morning secretion should be studied. The sediment should be collected in a conical glass, and in looking for tube-casts a large surface should be examined with a tolerably low power and little light. The arterio-sclerotic kidney may exist for a long time without the occurrence of albumen, or the albumen may be in very small quantities. In many cases it is impossible to differentiate the primary interstitial nephritis from an arterio-sclerotic kidney, nor clinically is it of any special value so to do. In persons under forty, with very high tension, great thickening of the superficial arteries, and marked hypertrophy of the heart, the renal are more likely to be secondary to the arterial changes.

Prognosis.—Chronic Bright's disease is an incurable affection, and the anatomical conditions on which it depends are quite as much beyond the reach of medicines as wrinkled skin or gray hair. Interstitial nephritis, however, is compatible with the enjoyment of life for many years, and it is now universally recognized that increased tension, thickening of the arterial walls, and polyuria with a small quantity of albumen, neither doom a man to death within a short time nor necessarily interfere with the pursuits of an active life so long as proper care be taken. I know patients who have had high tension and a little albumen in the urine with hyaline casts for ten, twelve, and, in one instance, fifteen years. Serious indications are the development of uræmic symptoms, dilatation of the heart, the onset of serous effusions, the development of Cheyne-Stokes breathing, persistent vomiting, and diarrhœa.

Treatment.—Patients without local indications or in whom the condition has been accidentally discovered should so regulate their lives as to throw the least possible strain upon heart, arteries, and kidneys. A quiet life without mental worry, with gentle but not excessive exercise, and residence in an equable climate, should be recommended. In addition they should be told to keep the bowels regular, the skin active by a daily tepid bath with friction, and the urinary secretion free by drinking daily a definite amount of either distilled water or some pleasant mineral water. Alcohol should be strictly prohibited. Tea and coffee are allowable.

The diet should be light and nourishing, and the patient should be warned not to eat excessively, and not to take meat more than once a day. Care in food and drink is probably the most important element in the treatment of these early cases.

A patient in good circumstances may be urged to go away during the winter months, or, if necessary, to move altogether to a warm equable cli-

mate, like that of southern California. There is no doubt of the value in these cases of removal from the changeable, irregular weather which prevails in the temperate regions from November until April.

At this period medicines are not required unless for certain special symptoms. Patients derive much benefit from an annual visit to certain mineral springs, such as Poland, Bedford, Saratoga, in this country, and Vichy and others in Europe. Mineral waters have no curative influence upon chronic Bright's disease; they simply help the interstitial circulation and keep the drains flushed. In this early stage, when the patient's condition is good, the tension not high, and the quantity of albumen small, medicines are not indicated, since no remedies are known to have the slightest influence upon the progress of the disease. Sooner or later symptoms arise which demand treatment. Of these the following are the most important:

(*a*) *Greatly Increased Arterial Tension.*—It is to be remembered that a certain increase of tension is not only necessary but unavoidable in chronic Bright's disease, and probably the most serious danger is too great lowering of the blood tension. The happy medium must be sought between such heightened tension as throws a serious strain upon the heart and risks rupture of the vessels and the low tension which, under these circumstances, is specially liable to be associated with serous effusions. In cases with persistent high tension the diet should be light, an occasional saline purge should be given, and sweating promoted by means of hot air or the hot bath. If these measures do not suffice, nitroglycerin may be tried, beginning with one minim of the one per cent. solution three times a day, and gradually increasing the dose if necessary. Patients vary so much in susceptibility to this drug that in each case it must be tested, the limit of dosage being that at which the patient experiences the physiological effect. As much as ten minims of the one per cent. solution may be given three times a day. In many cases I have given it in much larger doses for weeks at a time. I have never seen any ill effects from it. If the dose is excessive the patients complain at once of flushing or headache. Its use may be kept up for six or seven weeks, then stopped for a week and resumed. Its value is seen not only in the reduction of the tension, but also in the striking manner in which it relieves the headache, dizziness, and dyspnœa.

(*b*) More or less *anæmia* is present in advanced cases, which is best met by the use of iron. Weir Mitchell, who has had a unique experience in certain forms of chronic Bright's disease, gives the tincture of the perchloride of iron in large doses—from half a drachm to a drachm three times a day. He thinks that it not only benefits the anæmia, but that it also is an important means of reducing the arterial tension.

(*c*) Many patients with Bright's disease present themselves for treatment with signs of cardiac dilatation; there is a gallop rhythm or the heart sounds have a fœtal character, the breath is short, the urine scanty and

highly albuminous, and there are signs of local dropsy. In these cases the treatment must be directed to the heart. A morning dose of salts or calomel may be given, and digitalis in ten-minim doses, three or four times a day. Strychnia may be used with benefit in this condition. In some instances other cardiac tonics may be necessary, but as a rule the digitalis acts promptly and well.

(*d*) *Uræmic Symptoms.*—Even before marked manifestations are present there may be extreme restlessness, mental wandering, a heavy, foul breath, and a coated tongue. Headache is not often complained of, though intense frontal headache may be an early symptom of uræmia. In this condition, too, the patient may complain of palpitation, feelings of numbness, and sometimes nocturnal cramps. For these symptoms the saline purgatives should be ordered, and hot baths, so as to induce copious sweating. Nitroglycerin also may be freely used to reduce the tension. For the uræmic convulsions, if severe, inhalations of chloroform may be used. If the patient is robust and full-blooded, from twelve to twenty ounces of blood should be removed. The patient should be freely sweated, and if the convulsions tend to recur chloral may be given, either by the mouth or per rectum, or, better still, morphia. Uræmic coma must be treated by active purgation, and sweating should be promoted by the use of pilocarpine or the hot bath. For the restlessness and delirium morphia is indispensable. Since its recommendation in uræmic states some years ago, by Stephen MacKenzie, I have used this remedy extensively and can speak of its great value in these cases. I have never seen ill effects or any tendency to coma follow.

VII. AMYLOID DISEASE.

Amyloid (lardaceous or waxy) degeneration of the kidneys is simply an event in the process of chronic Bright's disease, most commonly in the chronic parenchymatous nephritis following fevers or of cachectic states. It has no claim to be regarded as one of the varieties of Bright's disease. The affection of the kidneys is generally a part of a wide-spread amyloid degeneration occurring in prolonged suppuration, as in disease of the bone, in syphilis, tuberculosis, and less commonly in association with leukæmia, lead poisoning, and gout.

Anatomically the amyloid kidney is large and pale, the surface smooth, and the venæ stellatæ well marked. On section the cortex is large and may show a peculiar glistening, infiltrated appearance, and the glomeruli are very distinct. The pyramids, in striking contrast to the cortex, are of a deep red color. A section soaked in dilute tincture of iodine shows spots of a walnut or mahogany brown color. The Malpighian tufts and the straight vessels may be most affected. In lardaceous disease of the kidneys the organs are not always enlarged. They may be normal in size or

small, pale, and granular. The amyloid change is first seen in the Malpighian tufts, and then involves the afferent and efferent vessels and the straight vessels. It may be confined entirely to them. In later stages of the disease the tubules are affected, chiefly the membrane, rarely, if ever, the cells themselves. In addition, the kidneys always show signs of diffuse nephritis. The Bowman's capsules are thickened, there may be glomerulitis, and the tubal epithelium is swollen, granular, and fatty.

Symptoms.—The renal features alone may not indicate the presence of this degeneration. Usually the associated condition gives a hint of the nature of the process. The urine, as a rule, shows important changes; the quantity is increased, and it is pale, clear, and of low specific gravity. The albumen is usually abundant, but it may be scanty, and in rare instances absent. Possibly the variations in the situation of the amyloid changes may account for this, since albumen is less likely to be present when the change is confined to the vasæ rectæ. In addition to ordinary albumen, globulin may be present. The tube-casts are variable, usually hyaline, often fatty or finely granular. Occasionally the amyloid reaction can be detected in the hyaline casts. Dropsy is present in many instances, particularly when there is much anæmia or profound cachexia. It is not, however, an invariable symptom, and there are cases in which it does not develop.

Increased arterial tension and cardiac hypertrophy are not usually present, except in those cases in which amyloid degeneration occurs in the secondary contracted kidney; under which circumstances there may be uræmia and retinal changes, which, as a rule, are not met with in other forms.

Diagnosis.—By the condition of the urine alone it is not possible to recognize amyloid changes in the kidney. Usually, however, there is no difficulty, since the Bright's disease comes on in association with syphilis, prolonged suppuration, disease of the bone, or tuberculosis, and there is evidence of enlargement of the liver and spleen. A suspicious circumstance is the existence of polyuria with a large amount of albumen in the urine, or when, in these constitutional affections, a large quantity of clear, pale urine is passed, even without the presence of albumen.

The prognosis depends rather on the condition with which the nephritis is associated. As a rule it is grave.

The treatment of the condition is that of chronic Bright's disease.

VIII. PYELITIS

(Consecutive Nephritis; Pyelonephritis; Pyonephrosis).

Definition.—Inflammation of the pelvis of the kidney and the conditions which result from it.

Etiology.—Pyelitis is induced by many causes, among which the following are the most important: (*a*) The irritation of calculi—a very

frequent cause. (*b*) Tubercle. (*c*) The infectious pyelitis which develops in typhoid fever, pneumonia, scarlet fever, diphtheria, small-pox, and other fevers. Here an acute inflammation of the pelvis of the kidney may occur, sometimes hæmorrhagic in character, more frequently diphtheritic. (*d*) The presence of decomposing urine, following pressure upon the ureter by tumors or bladder-disease. By far the most frequent form of pyelitis is that which is consecutive to cystitis, from whatever cause. In these cases the inflammation may not be confined to the pelvis, but pass to the kidney, inducing pyelonephritis. (*e*) Occasional causes are cancer, hydatids, the ova of certain parasites, and, according to some, the irritation of the saccharine urine of diabetes, and the irritation of turpentine or cubebs. (*f*) A primary pyelitis or pyelonephritis has been described as coming on after cold or overexertion, but such cases are extremely rare.

Morbid Anatomy.—In the early stages of pyelitis the mucous membrane is turbid, somewhat swollen, and may show ecchymoses. The urine in the pelvis is cloudy, and, on examination, numbers of epithelial cells are seen. In the form associated with the infectious fevers there is usually a grayish pseudo-membrane, either limited to an infundibulum or involving a great part of the pelvis.

In the calculous pyelitis there may be only slight turbidity of the membrane, which has been called by some catarrhal pyelitis. More commonly the mucosa is roughened, grayish in color, thick, and, on microscopical examination, the tissues are seen to be infiltrated with leucocytes. Under these circumstances there is almost always more or less dilatation of the calyces and flattening of the papillæ. Following this condition there may be (*a*) extension of the suppurative process to the kidney itself, forming a pyelonephritis; (*b*) a gradual dilatation of the calyces with atrophy of the kidney substance, and finally the production of the condition of pyonephrosis, in which the entire organ is represented by a sac of pus with or without a thin shell of renal tissue. (*c*) After the kidney structure has been destroyed by suppuration, and the obstruction at the orifice of the pelvis persists, the fluid portions may be absorbed, the pus becomes inspissated, so that the organ is represented by a series of sacculi containing grayish, putty-like masses, which may become impregnated with lime salts.

Tuberculous pyelitis, as already described, usually starts upon the apices of the pyramids, and may at first be limited in extent. Ultimately the condition produced may be similar to that of calculous pyelitis. Pyonephrosis is quite as frequent a sequence, while the final transformation of the pus into a putty-like material impregnated with salts, forming the so-called scrofulous kidney, is even commoner.

The pyelitis consecutive to cystitis is usually bilateral, and the kidney is apt to be involved, forming the so-called *surgical kidney*—acute suppurative nephritis. There are lines of suppuration extending along the pyramids, or small abscesses in the cortex, often just beneath the capsule;

or there may be wedge-shaped abscesses. The pus organisms either pass up the tubules or, as Steven has shown, pass by the lymphatics.

Symptoms.—The forms associated with the fevers rarely cause any symptoms, even when the process is extensive. In mild grades there is pain in the back or there may be tenderness on deep pressure on the affected side. The urine is turbid, contains a few mucous and pus cells, and occasionally blood-corpuscles. The urine is acid, and there may be a trace of albumen.

Before the condition of pyuria is established there may be attacks of pain on the affected side (not amounting to the severe agony of renal colic), rigors, high fever, and sweats. Under these circumstances the urine, which may have been clear, becomes turbid or smoky from the presence of blood, and may contain large numbers of mucus cells and transitional epithelium. These cases are not common, but I have twice had opportunity of studying such attacks for a prolonged period. In one patient the occurrence of the rigor and fever could sometimes be predicted from the change in the condition of the urine. Such cases occur, I believe, in association with calculi in the pelvis.

The statement is not infrequently made that the epithelium in the urine in pyelitis is distinctive and characteristic. This is erroneous, as may be readily demonstrated by comparing scrapings of the mucosa of the pelvis and of the bladder. In both the epithelium belongs to what is called the transitional variety, and in both regions the same conical, fusiform and irregular cells with long tails are found.

When the pyelitis, whether calculous or tuberculous, has become chronic and suppurative, the symptoms are:

(1) *Pyuria.*—The pus is in variable amount, and may be intermittent. Thus, as is often the case when only one kidney is involved, the ureter may be temporarily blocked, normal urine is passed for a time, and then there is a sudden outflow of the pent-up pus and the urine becomes purulent. Coincident with this retention, a tumor mass may be felt on the side affected. The pus has the ordinary characters, but the transitional epithelium is not so abundant at this stage and comes from the bladder or from the pelvis of the healthy side. Occasionally in rapidly advancing pyelonephritis portions of the kidney tissue, particularly of the apices of the pyramids, may slough away and appear in the urine; or, as in a remarkable specimen shown to me by Tyson, solid cheesy moulds of the calyces are passed. Casts from the kidney tubules are sometimes present. The reaction of the urine is at first acid, and may remain so even when the pus is passed in large quantities. If it remains any time in the bladder or if cystitis exists it becomes ammoniacal. Micturition may be very frequent and irritability of the bladder may be present.

(2) Intermittent fever associated with rigors is usually present in cases of suppurative pyelitis. The chills may recur at regular intervals, and the cases are often mistaken for malaria. Owen-Rees called attention to

the frequent occurrence of these rigors, which form a characteristic feature of both calculous and tuberculous pyelitis. Ultimately the fever assumes a hectic type and the rigors may cease.

(3) The general condition of the patient usually indicates prolonged suppuration. There is more or less wasting with anæmia and a progressive failure of health. Secondary abscesses may develop and the clinical picture becomes that of pyæmia. In some instances, particularly of tuberculous pyelitis, the clinical course may resemble that of typhoid fever. There are instances of pyuria recurring, at intervals, for many years without impairment of the bodily vigor.

(4) Physical examination in chronic pyelitis usually reveals tenderness on the affected side or a definite swelling, which may vary much in size and ultimately attain large dimensions if the kidney becomes enormously distended, as in pyonephrosis.

(5) Occasionally nervous symptoms, which may be associated with dyspnœa, supervene, or the termination may be by coma, not unlike that of diabetes. These have been attributed to the absorption of the decomposing materials in the urine, and has been called ammoniæmia. A form of paraplegia has been described in connection with some cases of abscess of the kidney, but whether due to a myelitis or to a peripheral neuritis has not yet been determined.

In suppurative nephritis or surgical kidney following cystitis, the patient complains of pain in the back, the fever becomes high, irregular, and associated with chills, and in acute cases a typhoid state develops in which death occurs.

Diagnosis.—Between the tuberculous and the calculous forms of pyelitis it may be difficult or impossible to distinguish, except by the detection of tubercle bacilli in the pus. This has been done on several occasions, but many slides must be examined, for the bacilli are usually scanty. From perinephric abscess pyonephrosis is distinguished by the more definite character of the tumor, the absence of œdematous swelling in the lumbar region, and, most important of all, the history of the case. The urine, too, in perinephric abscess may be free from pus. There are cases, however, in which it is difficult indeed to make a satisfactory diagnosis. A patient whom I saw with Fussell had had cystitis through her pregnancy, subsequently pus in the urine for several months, and then a large fluctuating abscess developed in the right lumbar region. It did not seem possible, either before or during the operation, to determine whether the case was a simple pyonephrosis or whether there had been a perinephric abscess caused by the pyelitis.

Suppurative pyelitis and cystitis are frequently confounded. I have known three instances of the former in which perineal section was performed on the supposition of the existence of an intractable cystitis. The two conditions may, of course, coexist and prove puzzling, but the history, the acid character of the pus in many instances, the less frequent

occurrence of ammoniacal decomposition, the local signs in one lumbar region, and the absence of pain in the bladder should be sufficient to differentiate the affections. In women, by catheterization of the ureters, it may be definitely determined whether the pus comes from the kidneys or from the bladder.

Prognosis.—Cases coming on during the fevers usually recover. Tuberculous pyelitis may terminate favorably by inspissation of the pus and conversion into a putty-like substance with deposition of lime salts. When pyonephrosis develops the dangers are increased. Perforation may occur, the patient may be worn out by the hectic fever, or amyloid disease may develop.

Treatment.—In mild cases fluids should be taken freely, particularly the alkaline mineral waters, to which the citrate of potash may be added.

The treatment of the calculous form will be considered later. Practically there are no remedies which have much influence upon the pyuria. Astringents in no way control the discharge, nor have I seen the slightest benefit from buchu, copaiba, sandal-wood oil, or uva ursi. Tonics should be given, a nourishing diet, and milk and butter-milk may be taken freely. When the tumor has formed or even before it is perceptible, if the symptoms are serious and severe, the kidney should be explored, and, if necessary, nephrotomy should be performed.

IX. HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis and calyces of the kidney with atrophy of its substance, caused by the accumulation of non-purulent fluids the result of obstruction.

Etiology.—The condition may be congenital, owing to some abnormality in the ureter or urethra. The tumor produced may be large enough to retard labor. Sometimes it is associated with other malformations. There is a condition of moderate dilatation, apparently congenital, which is not connected with any obstruction in the ducts. A case of the kind was shown at the Philadelphia Pathological Society by Daland.

In some instances there has been contraction or twisting of the ureter, or it has been inserted into the kidney at an acute angle or at a high level. In adult life the condition may be due to lodgement of a calculus, or to a cicatricial stricture following ulcer.

New growths, such as tubercle or cancer, occasionally induce hydronephrosis. More commonly, pressure upon the ureter from without, particularly tumors of the ovaries and uterus. Occasionally cicatricial bands compress the ureter. Obstruction within the bladder may result from cancer, from hypertrophy of the prostate with cystitis, and in the urethra from stricture. It is stated that slight grades of hydronephrosis have been found in patients with excessive polyuria.

In whatever way produced, when the ureter is blocked the secretion accumulates in the pelvis and infundibula. Sometimes acute inflammation follows, but more commonly the slow, gradual pressure causes atrophy of the papillæ with gradual distention and wasting of the organ. In acquired cases from pressure, even when dilatation is extreme, there may usually be seen a thin layer of renal structure. In the most extreme stages the kidney is represented by a large cyst, which may perhaps show on its inner surface imperfect septa. The fluid is thin and yellowish in color, and contains traces of urinary salts, urea, uric acid, and sometimes albumen. The secretion may be turbid from admixture with small quantities of pus.

Total occlusion does not always lead to a hydronephrosis, but may be followed by atrophy of the kidney. It appears that when the obstruction is intermittent or not complete the greatest dilatation is apt to follow. The sac may be enormous, and cause an abdominal tumor of the largest size. The condition has even been mistaken for ascites. Enlargement of the other kidney may compensate for the defect. Hypertrophy of the left side of the heart usually follows.

Symptoms.—When small, it may not be noticed. The congenital cases when bilateral usually prove fatal within a few days; when unilateral, the tumor may not be noticed for some time. It increases progressively and has all the characters of a tumor in the renal region. In adult life many of the cases, due to pressure by tumors, as in cancer of the uterus and enlargement of the prostate, etc., give rise to no symptoms.

There are remarkable instances of *intermittent* hydronephrosis in which the tumor suddenly disappears with the discharge of a large quantity of clear fluid. The sac gradually refills, and the process may be repeated for years. In these cases the obstruction is unilateral; a cicatricial stricture exists, or a valve is present in the ureter, or the ureter enters the upper part of the pelvis.

The examination of the abdomen shows, in unilateral hydronephrosis, a tumor occupying the renal region. When of moderate size it is readily recognized, but when large it may be confounded with ovarian or other tumors. In young children it may be mistaken for sarcoma of the kidney or of the retroperitoneal glands, the common causes of abdominal tumor in early life. Aspiration alone would enable us to differentiate between hydronephrosis and tumor. The large hydronephrotic sac is frequently mistaken for ovarian tumor. The latter is, as a rule, more mobile, and rarely fills the deeper portion of the lumbar region so thoroughly. The ascending colon can often be detected passing over the renal tumor, and examination per vaginam, particularly under ether, will give important indications as to the condition of the ovaries. In doubtful cases the sac should be aspirated. The fluid of the renal cyst is clear, or turbid from the presence of cell elements, rarely colloid in character; the specific gravity is low; albumen and traces of urea and uric acid are usually present; and the epithelial elements in it may be similar to those found in the pel-

vis of the kidney. In old sacs, however, the fluid may not be characteristic, since the urinary salts disappear, but in one case of several years' duration oxalate of lime and urea were found.

Perhaps the greatest difficulty is offered by the condition of hydronephrosis in a movable kidney. Here, the history of sudden disappearance of the tumor with the passage of a large quantity of clear fluid would be a point of great importance in the diagnosis. In those rare instances of an enormous sac filling the entire abdomen, and sometimes mistaken for ascites, the character of the fluid might be the only point of difference. The tumor of pyonephrosis may be practically the same in physical characteristics. Fever is usually present, and pus is often found in the urine. In these cases, when in doubt, exploratory puncture should be made.

The outlook in hydronephrosis depends much upon the cause. When single, the condition may never produce serious trouble, and the intermittent cases may persist for years. The latter are the most hopeful, and Frederick Taylor mentions an instance in which, after the fifth or sixth subsidence, in the course of two years, a calculus was discharged. Occasionally the cyst ruptures into the peritonæum, more rarely through the diaphragm into the lung. A remarkable case of this kind is at present under the care of my colleague, Halsted. A man, aged twenty-one, had, from his second year, attacks of abdominal pain in which a swelling would appear between the hip and costal margin and subside with the passage of a large amount of urine. In January, 1888, the sac discharged through the right lung.* Reaccumulations have occurred on several occasions since, and on June 9, 1891, the sac was opened and drained.

The sac may discharge spontaneously through the ureter and the fluid never reaccumulate. In bilateral hydronephrosis there is a danger that uræmia may supervene. There are instances, too, in which blocking of the ureter on the sound side by calculus has been followed by uræmia. And, lastly, the sac may suppurate, and the condition change to one of pyonephrosis.

Treatment.—Cases of intermittent hydronephrosis which do not cause serious symptoms should be let alone. It is stated that, in sacs of moderate size, the obstruction has been overcome by shampooing. If practised, it should be done with great care. When the sac reaches a large size aspiration may be performed and repeated if necessary. Puncture should be made in the flank, midway between the ilium and the last rib. If the fluid reaccumulates and the sac becomes large, it may be incised and drained, or, as a last resort, the kidney may be removed.

* Sowers, New York Medical Record, 1888.

X. NEPHROLITHIASIS (*Renal Calculus*).

Definition.—The formation in the kidney or in its pelvis of concretions, by the deposition of certain of the solid constituents of the urine.

Etiology and Pathology.—In the kidney substance itself the separation of the urinary salts produces a condition to which, unfortunately, the term infarct has been applied. Three varieties may be recognized: (1) The uric-acid infarct, usually met with at the apices of the pyramids in new-born children and during the first weeks of life. It is readily recognized as a yellowish linear streak in the pyramids and is of no significance; (2) the urate of soda infarct, sometimes associated with urate of ammonia, which forms whitish lines at the apices of the pyramids and is met with chiefly, but not always, in gouty persons; and (3) the lime infarcts, forming very opaque white lines in the pyramids, usually in old people.

In the pelvis and calyces concretions of the following forms occur: (*a*) Small gritty particles, *renal sand*, ranging in size from the individual grains of the uric-acid sediment to bodies one or two millimetres in diameter. These may be passed in the urine for long periods without producing any symptoms, since they are too fine to be arrested in their downward passage.

(*b*) Larger concretions, ranging in size from a small pea to a bean, and either solitary or multiple in the calyces and pelvis. It is the smaller of these calculi which, in their passage, produce the attacks of renal colic. They may be rounded and smooth, or present numerous irregular projections.

(*c*) The dendritic form of calculus. The orifice of the ureter may be blocked by a Y-shaped stone. The pelvis itself may be occupied by the concretion, which forms a more or less distinct mould. These are the remarkable *coral calculi*, which form in the pelvis complete moulds of infundibula and calyces, the latter even presenting cup-like depressions corresponding to the apices of the papillæ. Some of these casts in stone of the renal pelvis are as beautifully moulded as Hyrtl's corrosion preparations.

Chemically the varieties of calculi are: (1) Uric acid, by far the most important, which may form the renal sand, the small solitary, or the large dendritic stones. They are very hard, the surface is smooth, and the color reddish. The larger stones are usually stratified and very dense. Usually the uric acid and the urates are mixed, but in children stones composed of urates alone may occur.

(2) Oxalate of lime, which forms mulberry-shaped calculi, studded with points and spines. They are often very dark in color, intensely hard, and are a mixture of oxalate of lime and uric acid.

(3) Phosphatic calculi are composed of the phosphate of lime and the ammonio-magnesium phosphate, sometimes mixed with a small amount of

carbonate of lime. They are not common, since the phosphatic salts are oftener deposited about the uric acid or the oxalate of lime stones.

(4) Rare forms of calculi are made up of cystine, xanthine, carbonate of lime, indigo, and urostealith.

The mode of formation of calculi has been much discussed. They may be produced by an excess of a sparingly soluble abnormal ingredient, such as cystine or xanthine; more frequently by the presence of uric acid in a very acid urine which favors its deposition. Sir William Roberts thus briefly states the conditions which lead to the formation of the uric-acid concretions: high acidity, poverty in salines, low pigmentation, and high percentage of uric acid. The presence of albumen and mucus may determine, as Ord suggests, the deposition of the uric acid and thus form the starting point of a stone. Ova of parasites, blood-clot, casts, and shreds of epithelium may form the nuclei of stones.

Renal calculi are most common in the early and later periods of life. They are moderately frequent in this country, but there do not appear to be special districts, corresponding to the "stone counties" in England. Men are more often affected than women. Sedentary occupations seem to predispose to stone.

The effects of the calculi are varied. It is by no means uncommon to find a dozen or more stones of various sizes in the calyces without any destruction of the mucous membrane or dilatation of the pelvis. A turbid urine fills the pelvis in which there are numerous cells from the epithelial lining. There are cases of this sort in which, apparently, the stones may go on forming and are passed for years without seriously impairing the health and without inconvenience, except the attacks of renal colic. Still more remarkable are the cases of coral-like calculi, which may occupy the entire pelvis and calyces without causing pyelitis, but which gradually lead to more or less induration of the kidney. The most serious effects are when the stone excites a suppurative pyelitis and pyonephrosis.

Symptoms.—Patients may pass gravel for years without having an attack of renal colic, and a stone may never lodge in the ureter. In other instances, the formation of calculi goes on year by year and the patient has recurring attacks such as have been so graphically described by Montaigne in his own case. A patient may pass an enormous number of calculi. Some years ago I was consulted by a commercial traveller, an extremely vigorous man, who for many years had repeated attacks of renal colic, and had passed several hundred calculi of various sizes. His collection filled an ounce bottle. A patient may pass a single calculus, and never be troubled again. The large coral calculi may excite no symptoms. In a remarkable specimen of the kind, presented to the McGill Medical Museum by J. A. Macdonald, the patient, a middle-aged woman, died suddenly with uræmic symptoms. There was no pyelitis, but the kidneys were sclerotic.

Renal colic ensues when a stone enters the ureter. An attack may set in abruptly without apparent cause, or may follow a strain in lifting. It is characterized by agonizing pain, which starts in the flank of the affected side, passes down the ureter, and is felt in the testicle and along the inner side of the thigh. The pain may also radiate through the abdomen and chest, and be very intense in the back. In severe attacks there are nausea and vomiting and the patient is collapsed. The perspiration breaks out upon the face and the pulse is feeble and quick. A chill may precede the outbreak, and the temperature may rise as high 103°. No one has more graphically described an attack of "the stone" than Montaigne,* who was a sufferer for many years: "Thou art seen to sweat with pain, to look pale and red, to tremble, to vomit well-nigh to blood, to suffer strange contortions and convulsions, by starts to let tears drop from thine eyes, to urine thick, black, and frightful water, or to have it suppressed by some sharp and craggy stone, that cruelly pricks and tears thee." The symptoms persist for a variable period. In short attacks they do not last longer than an hour; in other instances they continue for a day or more, with temporary relief. Micturition is frequent, occasionally painful, and the urine, as a rule, is bloody. There are instances in which a large amount of clear urine is passed, probably from the other kidney. In rare cases the secretion of urine is completely suppressed, even when the kidney on the opposite side is normal, and death may occur from uræmia. This most frequently happens when the second kidney is extensively diseased, or when only a single kidney exists. A number of cases of this kind have been recorded. The condition has been termed, by Sir William Roberts, obstructive suppression. It is met with also when cancer compresses both ureters or involves their orifices in the bladder. The patient may not appear to be seriously ill at first, and uræmic symptoms may not develop for a week, when twitching of the muscles, great restlessness, and sometimes drowsiness supervene, but, strange to say, neither convulsions nor coma. Death takes place usually within twelve days from the onset of the obstruction.

After the attack of colic has passed there is more or less aching on the affected side, and the patient can usually tell from which kidney the stone has come. Examination during the attack is usually negative. Very rarely the kidney becomes palpable. Tenderness on the affected side is common. In very thin persons it may be possible, on examination of the abdomen, to feel the stone in the ureter; or the patient may complain of a grating sensation.

When the calculi remain in the kidney they may produce very definite and characteristic symptoms, of which the following are the most important:

(1) *Pain*, usually in the back, which is often no more than a dull sore-

* Essays, Book III, 13.

ness, but which may be severe and come on in paroxysms. It is usually on the side affected, but may be referred to the opposite kidney, and there are instances in which the pain has been confined to the sound side. Pains of a similar nature may occur in movable kidneys, and there are several instances on record in which surgeons have incised the kidney for stone and found none.

(2) *Hæmaturia.*—Although this occurs most frequently when the stone becomes engaged in the ureter, it may also come on when the stones are in the pelvis. The bleeding is seldom profuse, as in cancer, but in some instances may persist for a long time. It is aggravated by exertion and lessened by rest. Frequently it only gives to the urine a smoky hue. The urine may be free for days, and then a sudden exertion or a prolonged ride may cause smokiness, or blood may be passed in considerable quantities.

(3) *Pyelitis.*—(*a*) There may be attacks of severe pain in the back, not amounting to actual colic, which are initiated by a heavy chill followed by fever, in which the temperature may reach 104° or 105°, followed by profuse sweating. The urine, which has been clear, may become turbid and smoky and contain blood and abundant epithelium from the pelvis. Attacks of this description may recur at intervals for months or even years, and are generally mistaken for malaria, unless special attention is paid to the urine and to the existence of the pain in the back. This renal intermittent fever, due to the presence of calculi, is identical with the hepatic intermittent fever, due to gall-stones, and in both it is important to remember that the most intense paroxysms may occur without any evidence of suppuration.

(*b*) More frequently the symptoms of purulent pyelitis, which have already been described, are present; pain in the renal region, recurring chills, and pus in the urine, with or without indications of pyonephrosis.

(4) *Pyuria.*—There are instances of stone in the kidney in which pus occurs continuously or intermittently in the urine for many years. On many occasions between 1875 and 1884 I examined the urine of a physician who had passed calculi when a student in 1847, and has had pus in the urine at intervals ever since. There was no tumor. He had never had a second attack of colic. In spite of the prolonged suppuration he has had remarkable mental and bodily vigor.

Patients with stone in the kidney are often robust, high livers, and gouty. Attacks of dyspepsia are not uncommon, or they may have severe headaches.

Diagnosis.—Renal may be mistaken for intestinal colic, particularly if the distention of the bowels is marked, or for biliary colic. The situation and direction of the pain, the retraction and tenderness of the testicle, the occurrence of hæmaturia, and the altered character of the urine are distinctive features. Attention may again be called to the fact that attacks simulating renal colic are associated with movable kidney, or even, it has been supposed, without mobility of the kidney, with the accumu-

lation of the oxalates or uric acid in the pelvis of the kidney. The diagnosis between a stone in the kidney and stone in the bladder is not always easy, though in the latter the pain is particularly about the neck of the bladder, and not limited to one side. Important points are the reaction of the urine, which in stone in the bladder is almost invariably alkaline, and the abundance of mucus with the pus. It is stated that certain differences occur in the symptoms produced by different sorts of calculi. The large uric-acid calculi less frequently produce severe symptoms. On the other hand, as the oxalate of lime is a rougher calculus, it is apt to produce more pain (often of a radiating character) than the lithic-acid form, and to cause hæmorrhage. In both these forms the urine is acid. The phosphatic calculi are stated to produce the most intense pain, and the urine is commonly alkaline.

Treatment.—In the attacks of renal colic great relief is experienced by the hot bath, which is sometimes sufficient to relax the spasm. When the pain is very intense morphia should be given hypodermically, and inhalations of chloroform may be necessary until the effects of the anodyne are manifest. Local applications are sometimes grateful—hot poultices, or cloths wrung out of hot water. The patient may drink freely of hot lemonade, soda water, or barley water. Occasionally change in posture will give great relief, and inversion of the patient is said to be followed by immediate cessation of the pain.

In the intervals the patient should, as far as possible, live a quiet life, avoiding sudden exertion of all sorts. The essential feature in the treatment is to keep the urine abundant and, in a majority of the cases, alkaline. The patient should drink daily a large but definite quantity of mineral waters * or distilled water, which is just as satisfactory. The citrate or bicarbonate of potash may be added. The aching pains in the back are often greatly relieved by this treatment. Many patients find benefit from a stay at Saratoga, Bedford, Poland, or other mineral springs in this country, or at Vichy or Ems in Europe.

The diet should be carefully regulated, and similar to that indicated in the early stages of gout. Sir William Roberts recommends what is known as the solvent treatment for uric-acid calculi. The citrate of potash is given in large doses of half a drachm to a drachm every three hours in a tumblerful of water. This should be kept up for several months. I have had no success with this treatment, nor, when one considers the character of the uric-acid stones usually met with in the kidney, does it seem likely that any solvent action could be exercised upon them by changes in the urine. This treatment should be abandoned if the urine becomes ammoniacal.

The surgical treatment of stone in the kidney has advanced rapidly in the hands of Morris and others. It should be resorted to only when the

* Some of these, if we judge by the laudatory reports, are as potent as the waters of Corsena, declared by Montaigne to be "powerful enough to break stones."

attacks of pain are of such frequency as to interfere with the occupation of the patient, or when pyelitis or pyelonephritis has been excited. Stone in the kidney is not inconsistent with a long life and with the enjoyment of a fair measure of health.

XI. TUMORS OF THE KIDNEY.

These are benign and malignant. Of the benign tumors, the most common are the small nodular *fibromata* which occur frequently in the pyramids, the *aberrant adrenals* which Grawitz has described, and occasionally *lipoma*, *angioma*, or *lymphadenoma*. The *adenomata* may be congenital. In one of my cases the kidneys were greatly enlarged, contained small cysts, and numerous adenomatous structures throughout both organs.

Malignant growths—*cancer* or *sarcoma*—may be either primary or secondary. The sarcomata are the most common, either alveolar sarcoma or the remarkable form containing striped muscular fibres—rhabdo-myoma. Carcinoma is less frequent, and is of the encephaloid variety.

Primary cancer—meaning by this, malignant disease—is not uncommon, and the statistics given by some writers do not represent the frequency with which it is met with, at any rate, in this country. Virchow gives the ratio to cancer in other parts as one half of one per cent.

The tumors attain a very large size. In one of my cases the left kidney weighed twelve pounds and almost filled the abdomen. In children they may reach an enormous size. Morris states that in a boy at the Middlesex Hospital the tumor weighed thirty-one pounds. They grow rapidly, are often soft, and hæmorrhage frequently takes place into them. In the sarcomata invasion of the pelvis or of the renal vein is common. The rhabdo-myomas rarely form very large tumors, and death occurs shortly after birth. In one of my cases the child lived to the age of three years and a half. The tumor grew into the renal vein and inferior cava. A detached fragment passed as an embolus into the pulmonary artery, and a portion of it blocked the tricuspid orifice.

Symptoms.—The following are the most important: (1) Hæmaturia. This may be the first indication. The blood is fluid or clotted, and there may be very characteristic moulds of the pelvis of the kidney and of the ureter. It would no doubt be possible for such to form in the hæmaturia from calculus, but I have never met with a case of blood-casts of the pelvis and of the ureter, either alone or together, except in cancer. It is rare indeed that cancer elements may be recognized as in the urine. Of the numerous specimens which I have examined, in not one have I found elements which could be clearly distinguished from the multiform transitional epithelium constantly present in these cases.

(2) Pain is an uncertain symptom. In several of the largest tumors

which have come under my observation there has been no discomfort from beginning to close. When present, it is of a dragging, dull character, situated in the flank and radiating down the thigh. The passage of the clots may cause great pain

(3) Progressive emaciation. The loss of flesh is usually marked and advances rapidly. There may, however, be a very large tumor without emaciation.

Physical Signs.—In almost all instances tumor is present. When small and on the right side, it may be very movable; in some instances, occupying a position in the iliac fossa, it has been mistaken for ovarian tumor. The large growths fill the flank and gradually extend toward the middle line, occupying the right or left half of the abdomen. Inspection may show two or three hemispherical projections corresponding to distended sections of the organ. In children the abdomen may reach an enormous size and the veins are prominent and distended. On bimanual palpation the tumor is felt to occupy the lumbar region and can usually be lifted slightly from its bed; in some cases it is very movable, even when large; in others it is fixed, firm, and solid. The respiratory movements have but slight influence upon it. Rapidly growing renal tumors are soft, and on palpation may give a sense of fluctuation. A point of considerable importance is the fact that the colon crosses the tumor, and can usually be detected without difficulty.

Diagnosis.—In children very large abdominal tumors are either renal or retroperitoneal. The retroperitoneal sarcoma (Lobstein's cancer) is more central, but may attain as large a size. If the case is seen only toward the end, a differential diagnosis may be impossible; but as a rule the sarcoma is less movable. It is to be remembered that these tumors may invade the kidney. On the left side an enlarged spleen is readily distinguished, as the edge is very distinct and the notch or notches well marked; it descends during respiration, and the colon lies behind, not in front of it. On the right side growths of the liver are occasionally confounded with renal tumors; but such instances are rare, and there can usually be detected a zone of resonance between the upper margin of the renal tumor and the ribs. Late in the disease, however, this is not possible, for the renal tumor is in close union with the liver.

A malignant growth in a movable kidney may be very deceptive and may simulate cancer of the ovary or fibroid of the uterus. The great mobility upward of the renal growth and the negative result of examination of the pelvic viscera are the reliable points.

Medicinal treatment is of no avail. When the growth is small and the patient in good condition removal of the organ may be undertaken, but the percentage of cases of recovery is very small.

XII. CYSTIC DISEASE OF THE KIDNEY.

The following varieties of cysts are met with:

(1) The small cysts, already described in connection with the chronic nephritis, which result from dilatation of obstructed tubules or of Bowman's capsules.

(2) Solitary cysts, ranging in size from a marble to an orange, or even larger, are occasionally found in kidneys which present no other changes. They never give rise to symptoms, though, in exceptional cases, they may form tumors of considerable size. They, too, in all probability, result from obstruction.

(3) The congenital cystic kidneys. In this remarkable condition the kidneys are represented by a conglomeration of cysts, varying in size from a pea to a marble. The organs are greatly enlarged, and together may weigh six or more pounds. In the fœtus they may attain a size sufficient to impede labor. Little or no renal tissue may be noticeable, although in microscopical sections it is seen that a considerable amount remains in the interspaces. The cysts contain a clear or turbid fluid, sometimes reddish brown or even blackish in color, and may be of a colloidal consistence. Albumen, blood crystals, cholesterin, with triple phosphates and fat drops are found in the contents. Urea and uric acid are rarely present. The cysts are lined by a flattened epithelium. It is not yet accurately known how these cysts originate. That it is a defect in development rather than a pathological change is suggested by the fact that it is often in the embryo associated with other anomalies, particularly imperforate anus. Both Shattock and Bland Sutton, who have studied the question carefully, believe that the anomaly of development is in the failure of complete differentiation of the Wolffian bodies, which are, as it were, mixed with the kidneys and give rise to the cysts.

In a large majority of the cases death occurs, either *in utero* or shortly after birth; but instances are met with at all ages up to fifty or sixty, and I see no reason to suppose that these are not instances of persistence of the congenital form.

In the adult the tumors may be felt in the lumbar region as large rounded masses.

The *symptoms* are those of chronic interstitial nephritis. Many of the cases have presented no indications whatever until a sudden attack of uræmia; others have died of heart-failure. A rare termination in a case at the University Hospital, Philadelphia, was the rupture of one of the cysts and the production of a perinephritic abscess. The cardio-vascular changes induced are similar to those of interstitial nephritis. The left ventricle is hypertrophied and the arterial tension is greatly increased. The condition is compatible with excellent health. The dangers are those associated with chronic Bright's disease. It is important to remember that the conglomerate cystic kidney is almost invariably bilat-

eral. One kidney may be somewhat larger and more cystic than the other.

The diagnosis can sometimes be made. Great enlargement of both organs, with hypertrophy of the left heart and increased arterial tension, would suggest the condition.

Operative interference is not justifiable. I know an instance in which one kidney was removed and the patient died within twenty-four hours.

(4) Occasionally the kidneys and liver present numerous small cysts scattered through the substance. The spleen also may be involved. The cysts in the kidney are small, and neither so numerous nor so thickly set as in the conglomerate form, though in these cases the condition is probably the result of some congenital defect. There are cases, however, in which the kidneys are very large. It is more common in the lower animals than in man. I have seen several instances of it in the hog; in one case the liver weighed forty pounds, and was converted into a mass of simple cysts. The kidneys were less involved. Charles Kennedy * states that he has found references to twelve cases of combined cystic disease of the liver and kidneys.

The echinococcus cysts will be spoken of under the section on parasites.

XIII. PERINEPHRIC ABSCESS.

Suppuration in the connective tissue about the kidney may follow (1) blows and injuries; (2) the extension of inflammation from the pelvis of the kidney, the kidney itself, or the ureters; (3) perforation of the bowel, most commonly the appendix, in some instances the colon; (4) extension of suppuration from the spine, as in caries, or from the pleura, as in empyema; (5) as a sequel of the fevers, particularly in children.

In the post-mortem examination of a case of perinephric abscess the kidney is found surrounded by pus, particularly at the posterior part, though the pus may lie altogether in front, between the kidney and the peritonæum. Usually the abscess cavity is large and extensive. The pus is often offensive and may have a distinctly fæcal odor from contact with the large bowel. It may burrow in various directions and may burst into the pleura and be discharged through the lungs. A more frequent direction is down the psoas muscle, when it appears in the groin, or it may pass along the iliacus fascia and appear at Poupart's ligament. It may perforate the bowel or rupture into the peritonæum, and in some instances it has penetrated the bladder or vagina.

Post mortem we occasionally find a condition of chronic perinephritis in which the fatty capsule of the kidney is extremely firm, with numer-

* Laboratory Reports of the Royal College of Physicians, Edinburgh, vol. iii.

ous bands of fibrous tissue, and is stripped off from the proper capsule with the greatest difficulty. Such a condition probably produces no symptoms.

Symptoms.—There may be intense pain, aggravated by pressure, in the lumbar region. In other instances, the onset is insidious; there is no pain in the renal region, but on the first examination signs of deep-seated suppuration may be detected. On the affected side there is usually pain, which may be referred to the neighborhood of the hip-joint or radiate down the thigh and be associated with retraction of the testis. Sometimes the pain is referred even to the knee-joint, as in hip-disease. The patient lies with the thigh flexed, so as to relax the psoas muscle, and in walking throws, as far as possible, the weight on the opposite leg. According to Gibney, the patient keeps the spine immobile, assumes a stooping posture in walking, and has great difficulty in voluntarily adducting the thigh.

There may be pus in the urine if the disease has extended from the pelvis or the kidney, but in other forms the urine is clear. When pus has formed there are usually chills with irregular fever and sweats. On examination, deep-seated induration is felt between the last rib and the crest of the ilium. Bimanual palpation may reveal a distinct tumor or mass. Œdema or puffiness of the skin is frequently present.

The diagnosis of perinephric abscess is usually easy, and in any case when doubt exists the aspirator needle should be used. We cannot always differentiate the primary forms from those due to perforation of the kidney or of the bowel. This, however, makes but little difference, for the treatment is identical. It is usually possible by the history and examination to exclude disease of the vertebra. In children the condition is often mistaken for disease of the hip-joint, but the pain is higher, and there is an entire absence of fulness and tenderness over the hip-joint itself.

From whatever cause produced, the indications for treatment are identical—early, free, and permanent drainage.

SECTION VIII.

DISEASES OF THE NERVOUS SYSTEM.

I. DISEASES OF THE NERVES.

1. NEURITIS (*Inflammation of the Nerve Fibres*).

Neuritis may be *localized* in a single nerve, or *general*, involving a large number of nerves, in which case it is usually known as *multiple neuritis* or *polyneuritis*.

Etiology.—*Localized neuritis* arises from (*a*) cold, which is a very frequent cause, as, for example, in the facial nerve. This is sometimes known as rheumatic neuritis. (*b*) Traumatism—wounds, blows, direct pressure on the nerves, the tearing and stretching which follow a dislocation or a fracture, and the hypodermic injection of ether. Under this section come also the professional palsies, due to pressure in the exercise of certain occupations. (*c*) Extension of inflammation from neighboring parts, as in a neuritis of the facial nerve due to caries in the temporal bone, or in that met with in syphilitic disease of the bones, disease of the joints, and occasionally in tumors.

Multiple neuritis has a very complex etiology, the causes of which may be classified as follows: (*a*) The poisons of infectious diseases, as in leprosy, diphtheria, typhoid fever, small-pox, scarlet fever, and occasionally in other forms; (*b*) the organic poisons, comprising the diffusible stimulants, such as alcohol and ether, bisulphide of carbon, and naphtha, and the metallic bodies, such as lead, arsenic, and mercury; (*c*) cachectic conditions, such as occur in anæmia, cancer, tuberculosis, or marasmus from any cause; (*d*) the endemic neuritis or beri-beri; and (*e*) lastly, there are cases in which none of these factors prevail, but the disease sets in suddenly after overexertion or exposure to cold.

Morbid Anatomy.—In neuritis due to the extension of inflammation the nerve is usually swollen, infiltrated, and red in color. The inflammation may be chiefly perineural or it may pass into the deeper portion—*interstitial* neuritis—in which form there is an accumulation of lymphoid elements between the nerve bundles. The nerve fibres themselves may not appear involved, but there is an increase in the nuclei of

the sheath of Schwann. The myelin is fragmented, the nuclei of the internodal cells are swollen, and the axis cylinders present varicosities or undergo granular degeneration. Ultimately the nerve fibres may be completely destroyed and replaced by a fibrous connective tissue in which much fat is sometimes deposited—the *lipomatous neuritis* of Leyden.

In other instances the condition is termed *parenchymatous* neuritis, in which the changes are like those met with in the secondary or Wallerian degeneration, which follows when the nerve is cut off from its centre. The medullary substance and the axis cylinders are chiefly involved, the interstitial tissue being but little altered or only affected secondarily. The myelin becomes segmented and divides into small globules and granules, and the axis cylinders become granular, broken, subdivided, and ultimately disappear. The nuclei of the sheath of Schwann proliferate and ultimately the fibres are reduced to a state of atrophic tubes without a trace of the normal structure. The muscles connected with the degenerated nerves usually show marked atrophic changes, and in some instances the change in the nerve sheath appears to extend directly to the interstitial tissue of the muscles—the *neuritis fascians* of Eichhorst.

Symptoms. (*a*) **Localized Neuritis.**—As a rule the constitutional disturbances are slight. The most important symptom is pain of a boring or stabbing character, usually felt in the course of the nerve and in the parts to which it is distributed. The nerve itself is sensitive to pressure, probably, as Weir Mitchell suggests, owing to the irritation of its nervi nervorum. The skin may be slightly reddened or even œdematous over the seat of the inflammation. Mitchell has described increase in the temperature and sweating in the affected region, and such trophic disturbances as effusion into the joints and herpes. The function of the muscle to which the nerve fibres are distributed is impaired, motion is painful, and there may be twitchings or contractions. The tactile sensation of the part may be somewhat deadened, even when the pain is greatly increased. In the more chronic cases of local neuritis, such, for instance, as follow the dislocation of the humerus, the localized pain, which at first may be severe, gradually disappears, though some sensitiveness of the brachial plexus may persist for a long time, and the nerve cords may be felt to be swollen and firm. The pain is variable—sometimes intense and distressing; at others not causing much inconvenience. Numbness and formication may be present and the tactile sensation may be greatly impaired. The motor disturbances are marked. Ultimately there is extreme atrophy of the muscles. Contractures may occur in the fingers. The skin may be reddened or glossy, the subcutaneous tissue œdematous, and the nutrition of the nails may be defective.

A neuritis limited at first to a peripheral nerve may extend upward—the so-called ascending or migrating neuritis—and involve the larger nerve trunks, or even reach the spinal cord, causing subacute myelitis (Gowers). Thus, in a case reported by James Stewart, a girl of fourteen

had severe pain in the big toe of the left foot, which gradually extended up the leg and resisted all treatment until a portion of the sciatic nerve was removed. A year later she had pain in the little finger of the left hand, which gradually ascended along the ulnar nerve and required for its relief division and stretching. It has been suggested that the paralysis secondary to visceral disease, as of the bladder, may be due to an ascending neuritis. The inflammation may extend to the nerves of the other side, either through the spinal cord or its membranes, or without any involvement of the nerve centres, the so-called sympathetic neuritis. The electrical changes in localized neuritis vary a great deal, depending upon the extent to which the nerve is injured. The lesion may be so slight that the nerve and the muscles to which it is distributed may react normally to both currents; or it may be so severe that the typical reaction of degeneration develops within a few days, i. e., the nerve does not respond to stimulation by either current while the muscle reacts only to the galvanic current and in a peculiar manner. The contraction caused is slow and lazy, instead of sharp and quick as in the normal muscle, and the AnC contraction is usually stronger than the CC contraction. Between these two extremes there are many different grades and a careful electrical examination is most important as an aid to diagnosis and prognosis.*

The duration varies from a few days to weeks or months. A slight traumatic neuritis may pass off in a day or two, while the severer cases, such as follow unreduced dislocation of the humerus, may persist for months or never be completely relieved.

(*b*) **Multiple Neuritis.**—This presents a complex symptomatology. The following are the most important groups of cases:

(1) *Acute Febrile Polyneuritis.*—The attack follows exposure to cold or overexertion, or, in some instances, comes on spontaneously. The onset resembles that of an acute infectious disease. There may be a definite chill, pains in the back and limbs or joints, so that the case may be thought to be acute rheumatism. The temperature rises rapidly and may reach 103° or 104°. There are headache, loss of appetite, and the general symptoms of acute infection. The limbs and back ache. Intense pain in the nerves, however, is by no means constant. Tingling and formication are felt in the fingers and toes, and there is increased sensitiveness of the nerve trunks or of the entire limb. Loss of muscular power, first marked, perhaps, in the legs, gradually comes on and extends with the features of an ascending paralysis. In other cases the paralysis begins in the arms. The extensors of the wrists and the flexors of the ankles are early affected, so that there is foot and wrist drop. In severe cases there is general loss of muscular power, producing a flabby paralysis, which may extend to the muscles of the face and to the intercostals, and respiration may be carried on by the diaphragm alone. The muscles soften and waste rapidly. There

* See under facial paralysis.

may be only hyperæsthesia with soreness and stiffness of the limbs; in some cases, increased sensitiveness with anæsthesia; in other instances the sensory disturbances are slight. The clinical picture is not to be distinguished, in many cases, from Landry's paralysis; in others, from the subacute myelitis of Duchenne. James Ross concludes from an analysis of all the reported cases of the former disease that it coincides with multiple neuritis in general etiology, symptoms, and course. On the other hand, Hun, in a very thorough study of a recent case of Landry's paralysis, concludes that it is a separate and distinctive disease.

The course is variable. In the most intense forms the patient may die in a week or ten days, with involvement of the respiratory muscles or from paralysis of the heart. As a rule in cases of moderate severity, after persisting for five or six weeks, the condition remains stationary and then slow improvement begins. The paralysis in some muscles may persist for many months and contractures may occur from shortening of the muscles, but even when this occurs the outlook is, as a rule, good, although the paralysis may have lasted for a year or more.

(2) *Recurring Multiple Neuritis.*—Under the term *polyneuritis recurrens* Mary Sherwood has described from Eichhorst's clinic two cases in adults—in one case involving the nerves of the right arm, in the other both legs. In one patient there were three attacks, in the other two, the distribution in the various attacks being identical. There has recently been at my clinic a somewhat similar case—a man, aged thirty-one, who had, two and a half years ago, widespread paralysis, and who now has a second attack.

(3) *Alcoholic Neuritis.*—This, perhaps the most important form of multiple neuritis, was described in 1822 by James Jackson, Sr., of Boston, whose account of it is very graphic. Wilks recognized it as alcoholic paraplegia, but the starting point of the recent researches on the disease dates from the observation of Dumenil, of Rouen. Of late years our knowledge of the disease has extended rapidly, owing to the researches of Huss, Leyden, James Ross, Buzzard, and Henry Hun. It occurs most frequently in women, particularly steady, quiet tipplers. Its appearance may be the first revelation to the physician or to the family of habits of secret drinking. The onset is usually gradual, and may be preceded for weeks or months by neuralgic pains and tingling in the feet and hands. Convulsions are not uncommon. Fever is rare. The paralysis gradually sets in, at first in the feet and legs, and then in the hands and forearms. The extensors are affected more than the flexors, so that there is wrist-drop and foot-drop. The paralysis may be thus limited and not extend higher in the limbs. In other instances there is paraplegia alone, while in the most extreme cases all the extremities are involved. In rare instances the facial muscles and the sphincters are also affected. A case with this distribution recovered in my wards last year. The sensory symptoms are very variable. There are cases in which there are numbness and tingling only, without

great pain. In other cases there are severe burning or boring pains, the nerve trunks are sensitive, and the muscles are sore when grasped. The hands and feet are frequently swollen and congested, particularly when held down for a few moments. The cutaneous reflexes as a rule are preserved. The deep reflexes are usually lost.

The course of these alcoholic cases is, as a rule, favorable, and after persisting for weeks or months improvement gradually begins, the muscles regain their power, and even in the most desperate cases recovery may follow. The extensors of the feet may remain paralyzed for some time, and give to the patient a distinctive walk, the so-called *steppage* gait, characteristic of peripheral neuritis. It is sometimes known as the pseudo-tabetic gait, although in reality it could not well be mistaken for the gait of ataxia. The foot is thrown forcibly forward, the toe lifted high in the air so as not to trip upon it. The heel is brought down first and then the entire foot. It is an awkward, clumsy gait, and gives the patient the appearance of constantly stepping over obstacles. Among the most striking features of alcoholic neuritis are the mental symptoms. Delirium is common, and hallucinations with extravagant ideas, resembling somewhat those of general paralysis. In some cases the picture is that of ordinary delirium tremens, but the most peculiar and almost characteristic mental disorder is that so well described by Wilks, in which the patient loses all appreciation of time and place, and describes with circumstantial details long journeys which he has recently taken, or tells of persons whom he has just seen.

(4) *Multiple Neuritis in the Infectious Diseases.*—These have been already referred to, particularly in diphtheria, in which it is most common. The peripheral nature of the lesion in these instances has been shown by post-mortem examination. The outlook is usually favorable and, except in diphtheria, fatal cases are uncommon. Multiple neuritis in tuberculosis, diabetes, and syphilis is of the same nature, probably due to toxic materials absorbed into the blood.

(5) *Arsenical and Saturnine Neuritis.*—The arsenical neuritis is not common; only a single instance of it has come under my observation. No case to my knowledge has followed the use of Fowler's solution in my ward or dispensary practice, although I am in the habit of giving in chorea and anæmia doses which might be regarded as excessive. The most common causes are accidental poisoning, as in the cases reported by Mills. In a case of E. G. Cutler the patient got the arsenic from green-paper tags, which he was in the habit of putting in his mouth. The general symptoms are not unlike those of alcoholic paralysis; the weakness of the extensors is marked and the *steppage* gait characteristic. The neuritis due to lead will be discussed in the consideration of lead poisoning. The special involvement of the motor nerves and the great frequency of the occurrence of wrist-drop are the peculiarities of this form.

A similar form of neuritis is caused by the bisulphide of carbon.

(6) *Endemic Neuritis; Beri-beri.*—This is a widely spread disease in parts of India, and in China and Japan. To Shoubo and Baelz are due the credit of determining its true nature. It is probably due to a micro-organism. Food appears also to have a large share in its causation and it has been attributed to a fish diet. Some have thought it might be due to the presence of parasites in the intestines, but there are no grounds for this belief. There are several types of cases. In the acute pernicious form the nervous phenomena are not so marked. There are fever, anæmia, and general anasarca. In another group of cases there are numbness, loss of tendon reflexes, areas of anæsthesia, and muscular atrophy and anasarca. In other cases the paralysis and atrophy are the most prominent symptoms and the clinical picture is that of a rapidly progressing multiple neuritis with sensory and motor disturbances. The mortality varies from three or four to fifty per cent. Great difference of opinion still prevails concerning the cause of the disease. Special interest has been aroused in the subject in this country, owing to the fact that J. J. Putnam has described a similar disorder among the New England fishermen who frequent the Grand Banks. It occurs in epidemic form, and has, as prominent symptoms, general œdema, shortness of breath, and sensory disturbances with paralysis. In other instances, the paralysis is more extensive and proves fatal. In 1881 and 1889 there were epidemics among the crews of vessels fishing in this region. Birge describes eleven cases which occurred on one vessel in a crew of thirteen, two of whom died. One patient of this crew I saw with F. C. Shattuck, in the Massachusetts General Hospital, with the well-marked symptoms of multiple neuritis. The disease also exists in the West Indies, whence cases have come to this country (Seguin).

Diagnosis.—The electrical condition in multiple neuritis is thus described by Allen Starr: "The excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost and then the muscles contract to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and thus far it is quite pathognomonic of neuritis. For in anterior polio-myelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion.

"The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found and the negative pole produces stronger contractions than the positive pole. A loss of faradic irritability and a marked decrease in the galvanic irritability of

the muscle and nerve are therefore important symptoms of multiple neuritis." *.

There is rarely any difficulty in distinguishing the alcohol cases. The combination of wrist and foot drop with congestion of the hands and feet, and the peculiar delirium already referred to, is quite characteristic. The rapidly advancing cases with paralysis of all extremities, often reaching to the face and involving the sphincters, are more commonly regarded as of spinal origin, but the general opinion seems to point strongly to the fact that all such cases are peripheral. The less acute cases, in which the paralysis gradually involves the legs and arms with rapid wasting, simulate closely and are usually confounded with the subacute atrophic spinal paralysis of Duchenne. The diagnosis from locomotor ataxia is rarely difficult. The *steppage* gait is entirely different from that of tabes. There is rarely positive incoördination. The patient can usually stand well with the eyes closed. Foot-drop is not common in locomotor ataxia. The lightning pains are absent and there are no pupillary symptoms. The etiology, too, is of moment. The patient is recovering from a paralysis which has been more extensive, or from arsenical poisoning or has diabetes.

Treatment.—Rest in bed is essential. In the acute cases with fever, the salicylates and antipyrin are recommended. To allay the intense pain morphia or the hot applications of lead water and laudanum are often required. Great care must be exercised in treating the alcoholic form, and the attendant must not allow himself to be deceived by the statements of the relatives. It is sometimes exceedingly difficult to get a history of spirit-drinking. In the alcoholic form it is well to reduce the stimulants gradually. If there is any tendency to bed-sore an air-bed should be used or the patient placed in a continuous bath. Gentle friction of the muscles may be applied from the outset, and in the later stages, when the atrophy is marked and the pains have lessened, massage is probably the most reliable means at our command. Contractures may be gradually overcome by passive movements and extension. Often, with the most extreme deformity from contracture, recovery is, in time, still possible. The interrupted current is useful when the acute stage is passed.

Of internal remedies, strychnia is of value and may be given in increasing doses. Arsenic also may be employed, and if there is a history of syphilis the iodide of potassium and mercury may be given.

II. NEUROMATA.

Tumors situated on nerve fibres may consist of nerve substance proper, the true neuromata, or of fibrous tissue, the false neuromata. The true

* Lectures on Neuritis, Medical Record, New York, 1887.

neuroma usually contains nerve fibres only, or in rare instances ganglion cells. Cases of ganglionic or medullary neuroma are extremely rare; some of them, as Lancereaux suggests, are undoubtedly instances of malformation of the brain substance. In other instances, as in the case which I reported,* the tumor is, in all probability, a glioma with cells closely resembling those of the central nervous system. The true fascicular neuroma occurs in the form of the small subcutaneous painful tumor—*tubercula dolorosa*—which is situated on the nerves of the skin about the joints, sometimes on the face or on the breast. It is not always made up of nerve fibres, but may be, as shown by Hoggan, an adenomatous growth of the sweat glands.

The true neuromata, as a rule, are not painful, and occasionally are found associated with the nerve fibres in various regions. Those which develop at the ends and along the course of the nerves of the stump after amputation consist of connective tissue and of medullated and non-medullated nerve fibres. The most remarkable form is the *plexiform neuroma*, in which the various nerve cords are occupied by many hundreds of tumors. The cases are usually congenital. The tumors occur in all the nerves of the body. One of the most remarkable is that described by Prudden, the specimens of which are in the medical museum of Columbia College, New York. There were over 1,182 distinct tumors distributed on the nerves of the body. Prudden † has collected forty-one cases from the literature, in a majority of which the peripheral nerves were affected.

Neuromata rarely cause symptoms, except the subcutaneous painful tumor or those in the amputation stump. Here they may be very painful and cause great distress. Motor symptoms are sometimes present, particularly a constant twitching. Epilepsy has sometimes been associated, and relief has followed removal of the growths.

The only available treatment is excision. The subcutaneous painful tumor does not return, and excision completely relieves the symptoms. On the other hand, the amputation neuromata may recur.

III. DISEASES OF THE CRANIAL NERVES.

I. Olfactory Nerve.

The functions of this nerve may be disturbed at its peripheral ending, at the bulb, in the course of the nerve, or at the central origin in the brain. The disturbances may be manifested in subjective sensations of smell, complete loss of the sense, and occasionally in hyperæsthesia.

(*a*) *Subjective Sensations; Parosmia.*—Hallucinations of this kind are found in the insane and in epilepsy. The aura may be represented by an

* Journal of Anatomy and Physiology, vol. xv.

† American Journal of the Medical Sciences, vol. lxxx.

unpleasant odor, described as resembling chloride of lime, burning rags, or feathers. In a few cases with these subjective sensations tumors have been found in the hippocampal lobules. In rare instances, after injury of the head the sense is perverted—odors of the most different character may be alike, or the odor may be changed, as in a patient noted by Morell Mackenzie, who for some time could not touch cooked meat, as it smelt to her exactly like stinking fish.

(*b*) *Increased sensitiveness*, or *hyperosmia*, occurs chiefly in nervous, hysterical women, in whom it may sometimes be developed so greatly that, like a dog, they can recognize the difference between individuals by the odor alone.

(*c*) *Anosmia; Loss of the Sense of Smell.*—This may be produced by: (1) Affections of the termination of the nerve in the mucous membrane, which is perhaps the most frequent cause. It is by no means uncommon in association with chronic nasal catarrh and polypi. In paralysis of the fifth nerve, the sense of smell may be lost on the affected side, owing to interference with the secretion.

It is doubtful whether the cases of loss of smell following the inhalations of very foul or strong odors should come under this or under the central division.

(2) The lesions of the bulb or of the nerves. In falls or blows, in caries of the bones, and in meningitis or tumor, the bulbs or the nerve trunks may be involved. After an injury to the head the loss of smell may be the only symptom. Mackenzie notes a case of a surgeon who was thrown from his gig and lighted on his head. The injury was slight, but the anosmia which followed was persistent. In locomotor ataxia the sense of smell may be lost, due possibly to atrophy of the nerves.

(3) Lesions of the olfactory centre. There are congenital cases in which the nerve structures have not been developed. Cases have been reported by Beevor, Hughlings Jackson, and others, in which this symptom has been associated with disease in the hemisphere. The centre for the sense of smell is placed by Ferrier in the uncinate gyrus.

To test the sense of smell the pungent bodies, such as ammonia, which act upon the fifth nerve, should not be used, but such substances as cloves, peppermint, and musk. This sense is readily tested as a routine matter in brain cases by having two or three bottles containing the essential oils. In all instances a rhinoscopical examination should be made, as the condition may be due to local, not central causes. The *treatment* is unsatisfactory even in the cases due to local lesions in the nostrils.

II. Optic Nerve and Tract.

(1) *Lesions of the Retina.*

These are of importance to the physician, and information of the greatest value may be obtained by a systematic examination of the eye-

grounds. Only a brief reference can here be made to the more important of the appearances.

(*a*) **Retinitis.**—This occurs in certain general affections, more particularly in Bright's disease, syphilis, leukæmia, and anæmia. The common feature in all these states is the occurrence of hæmorrhage and the development of opacities. There may also be a diffuse cloudiness due to effusion of serum. The hæmorrhages are in the layer of nerve fibres. They vary greatly in size and form, but often follow the course of vessels. When recent the color is bright red, but they gradually change and old hæmorrhages are almost black. The white spots are due either to fibrinous exudate or to fatty degeneration of the retinal elements, and occasionally to accumulation of leucocytes or to a localized sclerosis of the retinal elements. The more important of the forms of retinitis to be recognized are:

Albuminuric retinitis, which occurs in chronic nephritis, particularly in the interstitial or contracted form. The percentage of cases affected is from fifteen to twenty-five. There are instances in which these retinal changes are associated with the granular kidney at a stage when the amount of albumen may be slight or transient; but in all such instances it will be found that there is a marked arterio-sclerosis. Gowers recognizes a degenerative form (most common), in which, with the retinal changes, there may be scarcely any alteration in the disk; a norrhagic form, with many hæmorrhages and but slight signs of infla on; and an inflammatory form, in which there is much swelling of the retina and obscuration of the disk. It is noteworthy that in some instances the inflammation of the optic nerve predominates over the other retinal changes and one may be in doubt for a time whether the condition is really associated with the renal changes or dependent upon intracranial disease.

Syphilitic Retinitis.—In the acquired form this is less common than choroiditis. In inherited syphilis *retinitis pigmentosa* is sometimes met with.

Retinitis in Anæmia.—It has long been known that a patient may become blind after a large hæmorrhage, either suddenly or within two or three days, and in one or both eyes. Occasionally the loss may be permanent and complete. In some of these instances a neuro-retinitis has been found, probably sufficient to account for the symptoms. In the more chronic anæmias, particularly in the pernicious form, retinitis is common, as determined first by Quincke.

In *malaria* retinitis or neuro-retinitis may be present, as noted by Stephen Mackenzie. It is seen only in the chronic cases with anæmia, and in my experience is not nearly so common proportionately as in pernicious anæmia. Of many instances which have come under my observation of severe malarial anæmia, particularly at the Philadelphia Hospital, there were only two with retinal hæmorrhages.

Leukæmic Retinitis.—In this affection the retinal veins are large

and distended; there is also a peculiar retinitis, as described by Liebreich. It is not very common. Of the seventeen cases of leukæmia which have come under my observation, retinitis existed in only three of the ten in which the eye-grounds were examined. There are numerous hæmorrhages and white or yellow areas, which may be large and prominent. In one of my cases the retina post mortem was occupied by many small, opaque, white spots, looking like little tumors, the larger of which had a diameter of nearly two millimetres. In Case 13 of my series the leukæmia was diagnosed by Norris and De Schweinitz, at whose clinic the patient had applied on account of failing vision, from the condition of the eye-grounds alone.

Retinitis is also found occasionally in diabetes, in purpura, in chronic lead poisoning, and sometimes as an idiopathic affection.

(*b*) **Functional Disturbances of the Retina.** (1) *Toxic Amaurosis.*—This occurs in uræmia and may follow convulsions or come on independently. Thus, a patient who had become suddenly blind the previous day, was led into one of my wards at the Montreal General Hospital. He had had no special symptoms, but examination showed extensive cardio-vascular changes. The urine was albuminous. The ophthalmoscopic examination was negative. The condition, as a rule, persists only for a day or two. This form of amaurosis occurs in poisoning by lead and occasionally by quinine. It seems more probable that the poisons act on the centres and not on the retina.

(2) *Hysterical Amaurosis.*—More frequently this is loss of acuteness of vision—amblyopia—but the loss of sight in one or both eyes may apparently be complete. The condition will be mentioned subsequently under hysteria.

(3) *Tobacco Amblyopia.*—The loss of sight is usually gradual, equal in both eyes, and affects particularly the centre of the field of vision. The eye-grounds may be normal, but occasionally there is congestion of the disks. On testing the color fields a central scotoma for red and green is found in all cases. Ultimately, if the use of tobacco is continued, organic changes may develop with atrophy of the disk.

(4) *Night-blindness—nyctalopia*—the condition in which objects are clearly seen during the day or by strong artificial light, but become invisible in the shade or in twilight, and *hemeralopia*, in which objects cannot be clearly seen without distress in daylight or in a strong artificial light, but are readily seen in a deep shade or in twilight, are functional anomalies of the retina which rarely come under the notice of the physician.

(5) *Retinal hyperæsthesia* is sometimes seen in hysterical women, but is not found frequently in actual retinitis. I have seen it once, however, in albuminuric retinitis and once, in a marked degree, in a patient with aortic insufficiency, in whose retinæ there were no signs other than the throbbing arteries.

(2) *Lesions of the Optic Nerve.*

(*a*) **Optic Neuritis** (*Papillitis*; *Choked Disk*).—In the first stage there is congestion of the disk and the edges are blurred and striated. In the second stage, the congestion is more marked, the swelling increases, the striation also is more visible. The physiological cupping disappears and hæmorrhages are not uncommon. The arteries present little change, the veins are dilated, and the disk may swell greatly. In slight grades of inflammation the swelling gradually subsides and occasionally the nerve recovers completely. In instances in which the swelling and exudate are very great, the subsidence is slow, and when it finally disappears there is complete atrophy of the nerve. The retina not infrequently participates in the inflammation, which is then a neuro-retinitis.

This condition is of the greatest importance in diagnosis. It may exist in its early stages without any disturbance of vision, and even with extensive papillitis the sight may for a time be good.

Optic neuritis is seen occasionally in anæmia and lead poisoning, more commonly in Bright's disease as neuro-retinitis. It occurs occasionally as a primary idiopathic affection. The frequent connection with intracranial disease, particularly tumor, makes its presence of great value to practitioners. The nature of the growth is without influence. In over ninety per cent of such instances the papillitis is bilateral. It is also found in meningitis, either the tuberculous or the simple form. In meningitis it is easy to see how the inflammation may extend down the nerve sheaths. In the case of tumor it was thought at first that a choked disk resulted from increased pressure within the skull. It is now more commonly regarded, however, as a descending neuritis.

(*b*) **Optic Atrophy.**—This may be: (1) A primary affection. Some of the cases have been hereditary and have come on in all the males of a family shortly after puberty. A large number of the cases of primary atrophy are associated with spinal disease, particularly locomotor ataxia. Other causes which have been assigned for the primary atrophy are cold, sexual excesses, diabetes, the specific fevers, alcohol, and lead.

(2) Secondary atrophy results from cortical lesions, pressure on the chiasma or on the nerves, or, most commonly of all, is a sequence of papillitis.

The ophthalmoscopic appearances are different in the cases of primary and secondary atrophy. In the former, the disk has a gray tint, the edges are well defined, and the arteries look almost normal; whereas in the consecutive atrophy the disk has a staring opaque-white aspect, with irregular outlines, and the arteries are very small.

The symptom of optic atrophy is loss of sight, proportionate to the damage in the nerve. The change is in three directions: "(1) Diminished acuity of vision; (2) alteration in the field of vision; and (3) altered perception of color." (Gowers.) The outlook in primary atrophy is bad; the

majority of cases go on to complete blindness. In the consecutive form there is greater chance of retention of slight vision.

(3) *Affections of the Chiasma and Tract.*

At the chiasma the optic nerves undergo partial decussation. Each optic tract, as it joins the chiasma, contains nerve fibres which supply half of the retina of either eye. Thus, of the fibres of the right tract, part pass the chiasma without decussating and supply the temporal half of the right retina, the other and larger portion of the fibres of the tract decussate in the chiasma and join the left optic nerve, supplying the nasal half of the retina on the other side. The fibres which cross are in the middle portion of the chiasma, while the direct fibres are on each side. The following are the most important changes which ensue in lesions of the tract and of the chiasma:

(*a*) *Unilateral Affection of Tract* (Fig. 1 *B*).—If right this produces loss of function in the temporal half of the retina on the right side, and on the nasal half of the retina on the left side, so that there is only half vision, and the patient is blind to objects on the left side. This is termed homonymous hemianopia or lateral hemianopia. The fibres passing to the right half of each retina being involved, necessarily the left half of each visual field is blind. The hemianopia may be partial and only a portion of the half field may be lost. The affected visual fields may have the normal extent, but in some instances there is considerable reduction. The color vision is, as a rule, lost in the half field—hemiachromatopia—but the half vision for color may be lost in central disease without any change in the field for white. When the left half of one field and the right half of the other, or vice versa, are blind, the condition is known as heteronymous hemianopia.

(*b*) *Disease of the Chiasma.*—(1) A lesion involves, as a rule, chiefly the central portion, in which the decussating fibres pass which supply the inner or nasal halves of the retinæ, producing in consequence loss of vision in the outer half of each field, or what is known as temporal hemianopia (Fig. 1 *H*).

(2) If the lesion is more extensive it may involve not only the central portion, but also the direct fibres on one side of the commissure, in which case there would be total blindness in one eye and temporal hemianopia in the other.

(3) Still more extensive disease is not infrequent from pressure of tumors in this region, the whole chiasma is involved, and total blindness results. The different stages in the process may often be traced in a single case from temporal hemianopia, then complete blindness in one eye with temporal hemianopia in the other, and finally complete blindness.

(4) A limited lesion of the outer part of the chiasma involves only the direct fibres passing to the temporal halves of the retinæ and inducing

blindness in the nasal field, or, as it is called, nasal hemianopia. This, of course, is extremely rare.

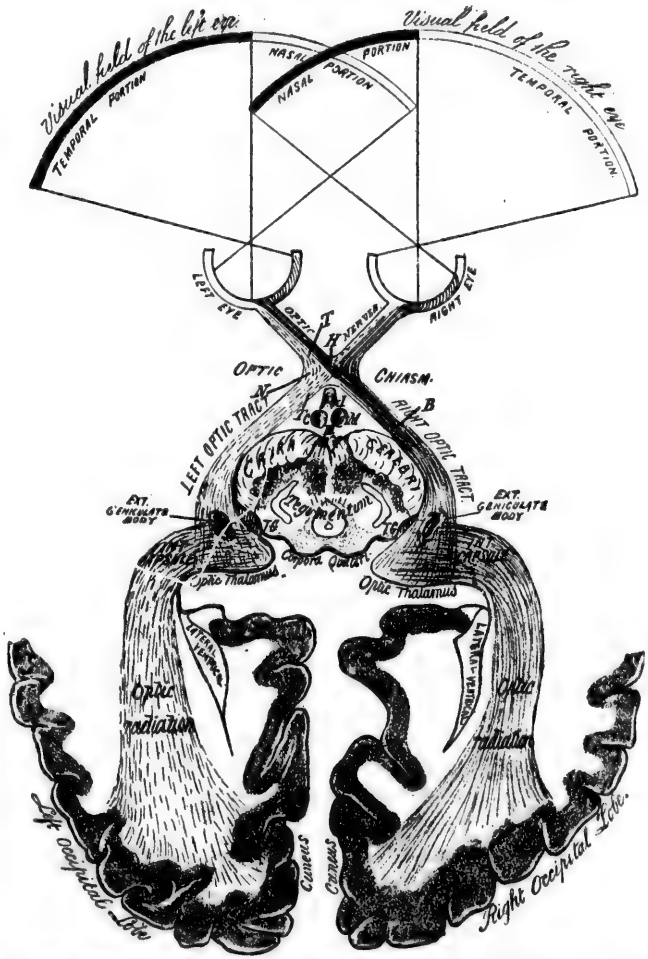


FIG. 1.—THE OPTIC AND VISUAL TRACTS (Starr). *N*, Lesion causing nasal hemianopia. *T*, Lesions causing temporal hemianopia. *H*, Lesion causing bilateral heteronymous hemianopia. *B*, Lesion of tract causing homonymous hemianopia.

(4) *Affections of the Tract and Centres.*

The optic tract crosses the crus to the hinder part of the optic thalamus and divides into two portions, one of which goes to the thalamus and external geniculate bodies and to the anterior quadrigeminal bodies. From

these parts fibres pass into the posterior part of the internal capsule and enter the occipital lobe, forming the fibres of the optic radiation (Fig. 1), which terminate in and about the cuneus, the region of the visual perceptive centre. The fibres of the other division of the tract pass to the internal geniculate bodies and to the posterior quadrigeminal body. It is still held by some physiologists that the cortical visual centre is not confined to the occipital lobe alone, but embraces the occipito-angular region.

A lesion of the fibres of the optic tract anywhere between the cortical centre and the chiasma will produce lateral hemianopia. The lesion may be situated: (*a*) In the tract itself. (*b*) In the region of the thalamus and the corpora quadrigemina, into which the larger part of each tract enters. (*c*) A lesion of the fibres passing from the corpora quadrigemina to the occipital lobe. This may be either in the hinder part of the internal capsule or the white fibres of the optic radiation. (*d*) Lesion of the cuneus. Bilateral disease of the cuneus may result in total blindness. (*e*) There is clinical evidence to show that lesion of the angular gyrus may be associated with visual defect, not so often hemianopia as crossed amblyopia, dimness of vision in the opposite eye, and great contraction in the field of vision. Lesions in this region are associated with mind blindness, a condition in which there is failure to recognize the nature of objects.

The effects of lesions in the optic nerve in different situations from the retinal expansion to the brain cortex are as follows: (1) Of the optic nerve —total blindness of the corresponding eye; (2) of the optic chiasma, either temporal hemianopia, if the central part alone is involved, or nasal hemianopia, if the lateral region of each chiasma is involved; (3) lesion of the optic tract between the chiasma and the geniculate bodies, produces lateral hemianopia; (4) lesion of the central fibres of the nerve between the geniculate bodies and the cerebral cortex produces lateral hemianopia; (5) lesion of the cuneus causes lateral hemianopia; and (6) lesion of the angular gyrus may be associated with hemianopia, sometimes crossed amblyopia, and the condition known as mind blindness.

Diagnosis.—The student or practitioner must have a clear idea of the physiology of the nerve centres before he can appreciate the symptoms or undertake the diagnosis of lesions of the optic nerve. Having determined the presence of hemianopia, the question arises as to the situation of the lesion, whether in the tract between the chiasma and the geniculate bodies or in the central portion of the fibres between these bodies and the visual centres. This can be determined in some cases by the test known as Wernicke's *hemiopic pupillary inaction*. The pupil reflex depends on the integrity of the retina or receiving membrane, on the fibres of the optic nerve and tract which transmit the impulse, and the nerve centre in the geniculate bodies which receives the impression and transmits it to the third nerve along which the motor impulses pass to the iris. If a bright

light is thrown into the eye and the pupil reacts, the integrity of this reflex arc is demonstrated. It is possible in cases of lateral hemianopia so to throw the light into the eye that it falls upon the blind half of the retina. If when this is done the pupil contracts, the indication is that the reflex arc above referred to is perfect, by which we mean that the optic nerve fibres from the retinal expansion to the centre, the centre itself, and the third nerve are uninvolved. In such a case the conclusion would be justified that the cause of the hemianopia was central; that is, situated behind the geniculate bodies, either in the fibres of the optic radiation or in the visual cortical centres. If, on the other hand, when the light is carefully thrown on the hemiopic half of the retina, the pupil remains inactive, the conclusion is justifiable that there is interruption in the path between the retina and the geniculate bodies, and that the hemianopia is not central, but dependent upon a lesion situated in the tract. This test of Wernicke's is sometimes difficult to obtain. It is best performed as follows: "The patient being in a dark or nearly dark room with the lamp or gas-light behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror held well out of focus upon the eye and note the size of the pupil. With my other hand I now throw a beam of light, focussed from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye; then laterally in various positions, and also from above and below the equator of the eye, noting the reaction at all angles of incidence of the ray of light." (Seguin.)

The significance of hemianopia varies. There is a functional hemianopia associated with migraine and hysteria. In a considerable proportion of all cases there are signs of organic brain-disease. Hemiplegia is common and the loss of power and blindness are on the same side. Thus, a lesion in the left hemisphere involving the motor tract produces right hemiplegia, and when the fibres of the optic radiation are involved in the internal capsule, there is also left lateral hemianopia, so that objects in the field of vision to the right are not perceived. Hemianæsthesia is not uncommon, owing to the close association of the sensory and visual tracts at the posterior part of the internal capsule. Certain forms of aphasia also occur in many of the cases.

III. Motor Nerves of the Eyeball.

Third Nerve.—Arising from the floor of the aqueduct of Sylvius, the nerve passes through the crus at the side of which it emerges. Passing along the wall of the cavernous sinus, it enters the orbit through the sphenoidal fissure and supplies, by its superior branch, the levator palpebræ superioris and the superior rectus, and by its inferior branch the in-

ternal and inferior recti muscles and the inferior oblique. It also supplies the ciliary muscle and the constrictor of the iris. Lesions may affect the centre or the nerve in its course and cause either paralysis or spasm.

Paralysis.—A nuclear lesion is usually associated with the disease of the centres for the other eye muscles, producing a condition of general ophthalmoplegia. More commonly the nerve itself is involved in its course, either by meningitis, gummata, or aneurism, or is attacked by neuritis, as in diphtheria and locomotor ataxia. Complete paralysis of the third nerve is accompanied by the following symptoms:

Paralysis of all the muscles, except the superior oblique and external rectus, by which the eye can be moved outward and a little downward and inward. There is divergent strabismus. There is ptosis or drooping of the upper eyelid, owing to paralysis of the levator palpebræ. The pupil is of medium size. It does not contract to light, and the power of accommodation is lost. The most striking features of this paralysis are the external strabismus, with diplopia or double vision, and the ptosis. In very many cases the affection of the third nerve is partial. Thus the levator palpebræ and the superior rectus may be involved together, or the ciliary muscles and the iris may be affected and the external muscles may escape.

There is a remarkable form of recurring oculo-motor paralysis affecting chiefly women, and involving all the branches of the nerve. In some cases the attacks have come on at intervals of a month; in others a much longer period has elapsed. The attacks may persist throughout life. They are sometimes associated with pain in the head and sometimes with migraine. Mary Sherwood has collected from the literature twenty-three cases.

Ptosis is a common and important symptom in nervous affections. We may here briefly refer to the conditions under which it may occur: (*a*) A congenital, incurable form, which is frequently seen; (*b*) the form associated with definite lesion of the third nerve, either in its course or at its nucleus. This may come on with paralysis of the superior rectus alone or with paralysis of the internal and inferior recti as well. (*c*) There are instances of complete or partial ptosis associated with cerebral lesions without any other branch of the third nerve being paralyzed. The position of the cortical centre is as yet unknown. (*d*) Hysterical ptosis, which is double and occurs with other hysterical symptoms. (*e*) Sympathetic or pseudo-ptosis is associated with symptoms of vaso-motor palsy, such as elevation of the temperature on the affected side with redness and œdema of the skin. Contraction of the pupil exists on the same side and the eyeball appears rather to have shrunk into the orbit. (*f*) In idiopathic muscular atrophy, when the face muscles are involved, there may be marked bilateral ptosis. And, lastly, in weak, deli-

cate women there is often to be seen a transient ptosis, particularly in the morning.

Among the most important of the symptoms of the third-nerve paralysis are those which relate to the ciliary muscle and iris.

Cycloplegia, paralysis of the ciliary muscle, causes loss of the power of accommodation. Distant vision is clear, but near objects cannot be properly seen. In consequence the vision is indistinct, but can be restored by the use of convex glasses. This may occur in one or in both eyes; in the latter case it is usually associated with disease in the nuclei of the nerve. Cycloplegia is an early and frequent symptom in diphtheritic paralysis and occurs also in tabes.

Iridoplegia, or paralysis of the iris, occurs in three forms (Gowers).

(*a*) *Accommodative iridoplegia*, in which the pupil does not diminish in size during the act of accommodation. To test for this the patient should look first at a distant and then at a near object in the same line of vision.

(*b*) *Reflex Iridoplegia.*—The path for the iris reflex is along the optic nerve and tract to the geniculate bodies, then to the nucleus of the third nerve, and along the trunk of this nerve to the ciliary ganglion, and so through the ciliary nerves to the eyes. Each eye should be tested separately, the other one being covered. The patient should look at a distant object in a dark part of the room; then a light is brought suddenly in front of the eye at a distance of three or four feet, so as to avoid the effect of accommodation. Loss of this iris reflex with retention of the accommodation contraction is known as the Argyll-Robertson pupil.

(*c*) *Loss of the Skin Reflex.*—If the skin of the neck is pinched or pricked the pupil dilates reflexly, the afferent impulses being conveyed along the cervical sympathetic. Erb pointed out that this skin reflex is lost usually in association with the reflex contraction, but the two are not necessarily conjoined. In iridoplegia the pupils are often small, particularly in spinal disease, as in the characteristic small pupils of tabes—spinal myosis. Iridoplegia may coexist with a pupil of medium size.

Inequality of the pupils—anisocoria—is not infrequent in progressive paresis and in tabes. It may also occur in perfectly healthy individuals.

Spasm.—Occasionally in meningitis and in hysteria there is spasm of the muscles supplied by the third nerve, particularly the internal rectus and the levator palpebræ. The clonic rhythmical spasm of the eye muscles is known as *nystagmus*, in which there is usually a bilateral, rhythmical, involuntary movement of the eyeballs. The condition is met with in many congenital and acquired brain lesions, in albinism, and sometimes in coal-miners.

Fourth Nerve.—This supplies the superior oblique muscle. In its course around the outer surface of the crus and in its passage into the

orbit it is liable to be compressed by tumors, by aneurism or in the exudation of basilar meningitis. Its nucleus in the upper part of the fourth ventricle may be involved by tumors or undergo degeneration with the other ocular nuclei. The superior oblique muscle acts in such a way as to direct the eyeball downward and rotates it slightly. The paralysis causes defective downward and inward movement, often too slight to be noticed. The head is inclined somewhat forward and toward the sound side, and there is double vision when the patient looks down, as in descending stairs.

Sixth Nerve.—This nerve emerges at the junction of the pons and medulla, then, passing forward, it enters the orbit and supplies the external rectus muscle. It is affected by meningitis at the base or by gummata or other tumors, and sometimes by cold. There is internal strabismus, and the eye cannot be turned outward. Diplopia occurs on looking toward the paralyzed side.

"When the nucleus is affected there is, in addition to paralysis of the external rectus, inability of the internal rectus of the opposite eye to turn that eye inwards. As a consequence of this the axes of the eyes are kept parallel and both are conjugately deviated to the opposite side, away from the side of lesion. The reason of this is that the nucleus of the sixth nerve sends fibres up in the pons to that part of the nucleus of the opposite third nerve which supplies the internal rectus. We thus have paralysis of the internal rectus without the nucleus of the third nerve being involved, owing to its receiving its nervous impulses for parallel movement from the sixth nucleus of the opposite side. As the sixth nucleus is in such proximity to the facial nerve in the substance of the pons, it is frequently found that the whole of the face on the same side is paralyzed, and gives the electrical reaction of degeneration, so that with a lesion of the *left* sixth nucleus there is conjugate deviation of both eyes to the *right*—i. e., paralysis of the left external and the right internal rectus, and sometimes complete paralysis of the *left* side of the face." (Beevor.)

General Features of Paralysis of the Motor Nerves of the Eye.—Gowers divides them into five groups:

(*a*) *Limitation of Movement.*—Thus, in paralysis of the external rectus, the eyeball is turned in by the contraction of the internal rectus and cannot be moved outward. When the paralysis is incomplete the movement is deficient in proportion to the degree of the palsy.

(*b*) *Strabismus.*—The axes of the eyes do not correspond. Thus, paralysis of the internal rectus causes a divergent squint; of the external rectus, a convergent squint. The deviation of the axis of the affected eye from parallelism with the other is called the primary deviation.

(*c*) *Secondary Deviation.*—If, while the patient is looking at an object, the sound eye is covered, so that he fixes the object looked at with the affected eye only, the sound eye is moved still further in the same di-

rection—e. g., outward—with paralysis of the opposite internal rectus. This is known as secondary deviation. It depends upon the fact that, if two muscles are acting together, when one is weak and an effort is made to contract it, the increased effort—innervation—acts powerfully upon the other muscle, causing an increased contraction.

(*d*) *Erroneous Projection.*—"We judge of the relation of external objects to each other by the relation of their images on the retina; but we judge of their relation to our own body by the position of the eyeball as indicated to us by the innervation we give to the ocular muscles" (Gowers). With the eyes at rest in the mid-position, an object at which we are looking is directly opposite our face. Turning the eyes to one side, we recognize that object in the middle of the field or to the side of this former position. We estimate the degree by the amount of movement of the eyes, and when the object moves and we follow it we judge of its position by the amount of movement of the eyeballs. When one ocular muscle is weak, the increased innervation gives the impression of a greater movement of the eye than has really taken place. The mind, at the same time, receives the idea that the object is further on one side than it really is, and in an attempt to touch it the finger may go beyond it. As the equilibrium of the body is in a large part maintained by a knowledge of the relation of external objects to it obtained by the action of the eye muscles, this erroneous projection resulting from paralysis disturbs the harmony of these visual impressions and may lead to giddiness—ocular vertigo.

(*e*) *Double Vision.*—This is one of the most disturbing features of paralysis of the eye muscles. The visual axes do not correspond, so that there is a double image—diplopia. That seen by the sound eye is termed the true image; that by the paralyzed eye, the false. In simple or homonymous diplopia the false image is "on the same side of the other as the eye by which it is seen." In crossed diplopia it is on the other side. In convergent squint the diplopia is simple; in divergent it is crossed.

Ophthalmoplegia.—Under this term is described a chronic progressive paralysis of the ocular muscles. Two forms are recognized—ophthalmoplegia *externa* and ophthalmoplegia *interna*. The conditions may occur separately or together and are described by Gowers under nuclear ocular palsy.

Ophthalmoplegia Externa.—The condition is one of more or less complete palsy of the external muscles of the eyeball, due usually to a slow degeneration in the nuclei of the nerves, but sometimes to pressure of tumors or to basilar meningitis. It is often but not necessarily associated with ophthalmoplegia interna. Siemerling, in the recent monograph in which he has analyzed the material (eight cases) left by the late Prof. Westphal, states that sixty-two cases are on record. In only eleven of these could syphilis be positively determined. The levator muscles of the eyelids and the superior recti are first involved, and gradually the other

muscles, so that the eyeballs are fixed and the eyelids droop. There is sometimes slight protrusion of the eyeballs. The disease is essentially chronic and may last for many years. It is found particularly in association with general paralysis, locomotor ataxia, and in progressive muscular atrophy. Mental disorders were present in eleven of the sixty-two cases. With it may be associated atrophy of the optic nerve and affections of other cranial nerves. Occasionally, as noted by Bristowe, it may be functional.

Ophthalmoplegia Interna.—Jonathan Hutchinson applied this term to a progressive paralysis of the internal ocular muscles, causing loss of pupillary action and the power of accommodation. When the internal and external muscles are involved the affection is known as total ophthalmoplegia, and in a majority of the cases the two conditions are associated. In some instances the internal form may depend upon disease of the ciliary ganglion.

While, as a rule, ophthalmoplegia is a chronic process, there is an acute form associated with hæmorrhagic softening of the nuclei of the ocular muscles. There is usually marked cerebral disturbance. It was to this form that Wernicke gave the name polio-encephalitis superior.

Treatment of Ocular Palsies.—It is important to ascertain, if possible, the cause. The forms associated with locomotor ataxia are obstinate, and resist treatment. Occasionally, however, a palsy, complete or partial, may pass away spontaneously. The group of cases associated with chronic degenerative changes, as in progressive paresis and bulbar paralysis, is little affected by treatment. On the other hand, in syphilitic cases, mercury and iodide of potassium are indicated and are often beneficial. Arsenic and strychnia, the latter hypodermically, may be employed. In any case in which the onset is acute, with pain, hot fomentations and counter-irritation or leeches applied to the temple give relief. The direct treatment by electricity has been extensively employed, but probably without any special effect. The diplopia may be relieved by the use of prisms, or it may be necessary to cover the affected eye with an opaque glass.

IV. Fifth Nerve.

Paralysis may result from: (*a*) Disease of the pons, particularly hæmorrhage or patches of sclerosis. (*b*) Injury or disease at the base of the brain. Fracture rarely involves the nerve; on the other hand, meningitis, acute or chronic, and caries of the bone are not uncommon causes. (*c*) The branches may be affected as they pass out—the first division by tumors pressing on the cavernous sinus or by aneurism; the second and third divisions by growths which invade the spheno-maxillary fossa. (*d*) Primary neuritis, which is rare.

Symptoms.—(*a*) *Sensory Portion.*—Paralysis of the fifth nerve causes loss of sensation in the parts supplied, including the half of the

face, the corresponding side of the head, the conjunctiva, the mucosa of the lips, tongue, hard and soft palate, and of the nose of the same side. The anæsthesia may be preceded by tingling or pain. The muscles of the face are also insensible and the movements may be slower. The sense of smell is interfered with. There is loss of the sense of taste. There are, in addition, *trophic* changes; the salivary, lachrymal, and buccal secretions may be lessened, abrasions of the mucous membranes heal slowly, and the teeth may become loose. The eye inflames, the corneæ become cloudy and may ulcerate. These latter symptoms occur only when the Gasserian ganglion is affected, as the nerve itself may be involved for years without producing ophthalmia. Herpes may develop in the region supplied by the nerve and is usually associated with much pain. It is most common in the upper branch of the nerve. The pain which follows the herpes may be peculiarly enduring, lasting for months or years (Gowers).

(*b*) *Motor Portion.*—The inability to use the muscles of mastication on the affected side is the distinguishing feature of paralysis of this portion of the nerve. It is recognized by placing the finger on the masseter and temporal muscles, and, when the patient closes the jaw, the feebleness of their contraction is noted. If paralyzed, the external pterygoid cannot move the jaw toward the unaffected side; and when depressed, the jaw deviates to the paralyzed side. The motor paralysis of the fifth nerve is almost invariably a result of involvement of the nerve after it has left the nucleus. Cases, however, have been associated with cortical lesions. Hirt concludes, from his case, that the motor centre for the trigeminus is in the neighborhood of the lower third of the ascending frontal convolution.

Spasm of the Muscles of Mastication.—Trismus, the masticatory spasm of Romberg, may be tonic or clonic, and is either an associated phenomenon in general convulsions or, more rarely, an independent affection. In the tonic form the jaws are kept close together—lock-jaw—or can be separated only for a short space. The muscles of mastication can be seen in contraction and felt to be hard and the spasm is often painful. This tonic contraction is an early symptom in tetanus, and is sometimes seen in tetany. A form of this tonic spasm occurs in hysteria. Occasionally trismus follows exposure to cold, and is said to be due to reflex irritation from the teeth, the mouth, or caries of the jaw. It may also be a symptom of organic disease due to irritation near the motor nucleus of the fifth nerve.

Clonic spasm of the muscles supplied by the fifth occurs in the form of rapidly repeated contractions, as in "chattering teeth." This is rare apart from general conditions, though cases are on record, usually in women late in life, in whom this isolated clonic spasm of the muscles of the jaw has been found. In another form of clonic spasm sometimes seen in chorea, there are forcible single contractions. Gowers mentions an instance of its occurrence as an isolated affection.

(*c*) *Gustatory.*—Loss of the sense of taste in the anterior two thirds of

the tongue, as a rule, follows paralysis of the fifth nerve. The gustatory fibres pass from the chorda tympani to the lingual branch of the fifth. Disease of the fifth nerve is, however, not always associated with loss of taste in the anterior part of the tongue, in which case either the taste fibres escape, or the disease is within the pons where these fibres are separate from those of sensation.

The *diagnosis* of disease of the trifacial nerve is rarely difficult. It must be remembered that the preliminary pain and hyperæsthesia are sometimes mistaken for neuralgia. The loss of sensation and the palsy of the muscles of mastication are readily determined.

Treatment.—When the pain is severe morphia may be required and local applications are useful. If there is a suspicion of syphilis, appropriate treatment should be given. Faradization is sometimes beneficial.

V. Facial Nerve.

Paralysis (*Bell's Palsy*).—The *portio dura* of the seventh pair may be paralyzed by (1) lesions of the cortex—supranuclear palsy; (2) lesions of the nucleus itself; or (3) involvement of the nerve trunk in its tortuous course within the pons and through the wall of the skull.

I. *Supranuclear Paralysis*, due to lesion of the cortex or of the facial fibres in the corona radiata or internal capsule, is, as a rule, associated with hemiplegia. It may be caused by tumors, abscess, chronic inflammation, or softening in the region of the internal capsule. It is distinguished from the peripheral form by two well-marked characters—the persistence of the normal electrical excitability of both nerves and muscles and the absence of involvement of the upper branches of the nerve, so that the orbicularis palpebrarum and frontalis muscle are spared. A third difference is that in this form the voluntary movements are more impaired than the emotional. There are instances of cortical facial paralysis—monoplegia facialis—associated with lesions in the centre for the face muscles in the lower Rolandic region. Isolated paralysis, due to involvement of the nerve fibres in their path to the nucleus, is uncommon. In the great majority of cases supranuclear facial paralysis is part of a hemiplegia. Paralysis is on the same side as that of the arm and leg because the facial muscles bear precisely the same relation to the cortex as the spinal muscles. The nuclei of origin on either side of the middle line in the medulla are united by decussating fibres with the cortical centre on the opposite side (see Fig. 3).

II. The *nuclear paralysis* caused by lesions of the nerve centre in the medulla is not common alone; but is seen occasionally in tumors, chronic softening, and hæmorrhage. In rare instances of anterior polio-myelitis the facial nucleus is affected. In diphtheria this centre may also be involved. The symptoms are practically similar to those of an affection of the nerve fibre itself—infranuclear paralysis.

III. *Involvement of the Nerve Trunk.*—Paralysis may result from:

(*a*) Involvement of the nerve as it passes through the pons—that is, between its nucleus in the floor of the fourth ventricle and the point of emergence in the postero-lateral aspect of the pons. The specially interesting feature in connection with involvement of this part is the production of what is called alternating or *cross paralysis*, the face being involved on the same side as the lesion, and the arm and leg on the opposite side, since the motor path is involved above the point of decussation in the medulla (Fig. 3, *z*). This occurs only when the lesion is in the lower section of the pons. A lesion in the upper division involves the fibres not of the outgoing nerve on the same side, but of the nerve of the other side, which has crossed and is ascending to the hemisphere. In this case there would of course be, as in hemiplegia, paralysis of the face and limbs on the side opposite to the lesion. The palsy, too, would resemble the cerebral form, involving only the lower fibres of the facial nerve.

(*b*) The nerve may be involved at its point of emergence by tumors, gummata, meningitis, or occasionally may be injured in fracture of the base.

(*c*) In passing through the Fallopian canal the nerve may be involved in disease of the ear, particularly by caries of the bone in otitis media. This is a common cause in children.

(*d*) As the nerve emerges from the styloid foramen it is exposed to injuries and blows which not infrequently cause paralysis. The fibres may be cut in the removal of tumors in this region, or the paralysis may be caused by pressure of the forceps in an instrumental delivery.

(*e*) Exposure to cold is the most common cause of facial paralysis, inducing a neuritis of the nerve within the Fallopian canal. It is sometimes termed rheumatic neuritis, but there is no evidence that it is specially associated with the rheumatic poison.

Facial diplegia is a rare condition occasionally found in affections at the base of the brain, lesions in the pons, simultaneous involvement of the nerves in ear disease, and in diphtheritic paralysis. Disease of the nuclei or symmetrical involvement of the cortex might also produce it.

Symptoms.—In the peripheral facial paralysis all the branches of the nerve are involved. The face on the affected side is immobile and can neither be moved at will nor participate in any emotional movements. The skin is smooth and the wrinkles are effaced, a point particularly noticeable on the forehead of elderly persons. The eye cannot be closed, the lower lid droops, and the eye waters. On the affected side the angle of the mouth is lowered, and in drinking the lips are not kept in close apposition to the glass, so that the liquid is apt to run out. In smiling or laughing the contrast is most striking, as the affected side does not move, which gives a curious unequal appearance to the two sides of the face. The eye cannot be closed and the forehead cannot be wrinkled. On asking a patient to show his upper teeth, the angle of the mouth is not raised. In

all these movements the face is drawn to the sound side by the action of the muscles. Speaking may be slightly interfered with, owing to the imperfection in the formation of the labial sounds. Whistling cannot be performed. In chewing the food, owing to the paralysis of the buccinator, particles collect on the affected side. The paralysis of the nasal muscles is seen on asking the patient to sniff. Owing to the fact that the lips are drawn to the sound side, the tongue, when protruded, looks as if it were pushed to the paralyzed side; but on taking its position from the incisor teeth, it will be found to be in the middle line. The reflex movements are lost in this peripheral form. It is usually stated that the palate is paralyzed on the same side and that the uvula deviates. Both Gowers and Hughlings Jackson deny the existence of this involvement in the great majority of cases, and Horsley and Beevor have shown that these parts are innervated by the accessory nerve to the vagus.

When the nerve is involved within the canal between the genu and the origin of the chorda tympani, the sense of taste may be lost in the anterior part of the tongue on the affected side. When the nerve is damaged outside the skull the sense of taste is unaffected. Hearing is often impaired in facial paralysis, most commonly by preceding ear disease. The paralysis of the stapedius muscle may lead to increased sensitiveness to musical notes. Herpes is sometimes associated with facial paralysis. Pain is not common, but there may be neuralgia about the ear.

The *electrical reactions*, which are those of a peripheral palsy, have considerable importance from a prognostic standpoint. Erb's rules are as follows: If there is no change, either faradic or galvanic, the prognosis is good and recovery takes place in from fourteen to twenty days. If the faradic and galvanic excitability of the nerve is only lessened and that of the muscle increased to the galvanic current and the contraction formula altered (the contraction sluggish AnC>CC), the outlook is relatively good and recovery will probably take place in from four to six weeks; occasionally in from eight to ten. When the reaction of degeneration is present—that is, if the faradic and galvanic excitability of the nerves and the faradic excitability of the muscles are lost and the galvanic excitability of the muscle is quantitatively increased and qualitatively changed, and if the mechanical excitability is altered—the prognosis is relatively unfavorable and the recovery may not occur for two, six, eight, or even fifteen months.

The *course* of facial paralysis is usually favorable. The onset in the form following cold is very rapid, developing perhaps within twenty-four hours, but rarely is the paralysis permanent. On the other hand, in the paralysis from injury, as by a blow on the mastoid process, the paralysis may remain. When permanent the muscles are entirely toneless. In some instances contracture develops as the voluntary power returns, and the natural folds and the wrinkles on the affected side may be deepened, so that on

looking at the face one at first may have the impression that the affected side is the sound one. This is corrected at once on asking the patient to smile, when it is seen which side of the face has the most active movement.

The *diagnosis* of facial paralysis is usually easy. The distinction between peripheral and central is based on facts already mentioned.

Treatment.—In the cases which result from cold and are probably due to neuritis within the bony canal, hot applications first should be made; subsequently the thermo-cautery may be used lightly at intervals of a day or two over the mastoid process, or small blisters applied. If the ear is diseased, free discharge for the secretion should be obtained. The continuous current may be employed to keep up the nutrition of the muscles. The positive pole should be placed behind the ear, the negative one along the zygomatic and other muscles. The application can be made daily for a quarter of an hour and the patient can readily be taught to make it himself before the looking-glass. Massage of the muscles of the face is also useful.

A course of iodide of potassium may be given even when there is no indication of syphilis.

Spasm.—The spasm may be limited to a few or involve all the muscles innervated by the facial nerve and may be unilateral or bilateral.

It is known also by the name of mimic spasm or of convulsive tic. Several different affections are usually considered under the name of facial or mimic spasm, but we shall here speak only of the simple spasm of the facial muscles, either primary or following paralysis, and shall not include the cases of habit spasm in children, or the *tic convulsif* of the French.

Gowers recognizes two classes—one in which there is an organic lesion, and an idiopathic form. It is thought to be due also to reflex causes, such as the irritation from carious teeth or the presence of intestinal worms. The disease usually occurs in adults, whereas the habit spasm and the *tic convulsif* of the French, often confounded with it, are most common in children. True mimic spasm occasionally comes on in childhood and persists. In the case of a school-mate, the affection was marked as early as the eleventh or twelfth year and still continues. When the result of organic disease there has usually been a lesion of the centre in the cortex, as in the case reported by Berkeley, or pressure on the nerve at the base of the brain by aneurism or tumor.

Symptoms.—The spasm may involve only the muscles around the eye—blepharospasm—in which case there is constant, rapid, quick action of the orbicularis palpebrarum, which, in association with photophobia, may be tonic in character. More commonly the spasm affects the lateral facial muscles with those of the eye and there is constant twitching of the side of the face with partial closure of the eye. The frontalis is rarely in-

volved. In aggravated cases the depressors of the angle of the mouth, the levator menti, and the platysma myoides are affected. This spasm is confined to one side of the face in a majority of cases, though it may extend and become bilateral. It is increased by emotional causes and involuntary movements of the face. As a rule, it is painless, but there may be tender points on the course of the fifth nerve, particularly the supraorbital branch. Tonic spasm of the facial muscle may follow paralysis, and is said to result occasionally from cold.

The outlook in facial spasm is always dubious. A majority of the cases persist for years and are incurable.

Treatment.—Sources of irritation should be looked for and removed. When a painful spot is present over the fifth nerve, blistering or the application of the thermo-cautery may relieve it. Hypodermic injections of strychnia may be tried, but are of doubtful benefit. Weir Mitchell recommends the freezing of the cheek for a few minutes daily or every second day with the spray, and this, in some instances, is beneficial. Often the relief is transient; the cases return, and at every clinic may be seen half a dozen or more of such patients who have run the gamut of all measures without material improvement. Operative interference may be resorted to in severe cases, although not much can be expected of it.

VI. Auditory Nerve.

This nerve, forming the *portio mollis* of the seventh pair, enters the internal auditory meatus, and divides into the cochlear and vestibular branches. The cortical centre for hearing is in the temporo-sphenoidal lobe. Primary disease of the auditory nerve in its centre or intracranial course is uncommon. More frequently the terminal branches are affected within the labyrinth.

(*a*) *Affection of the Cortical Centre.*—In the monkey, experiments indicate that the first temporal gyri represent the centre for hearing. In man the cases of disease indicate that it has the same situation, as destruction of this gyrus on the left side results in word-deafness, which may be defined as an inability to understand the meaning of words, though they may still be heard as sounds. The central fibres of the auditory nerve between the cortical centre and the nucleus in the fourth ventricle may be involved and produce deafness. This has resulted from the presence of a tumor in the corpora quadrigemina, and may be associated with a lesion of the internal capsule.

(*b*) *Lesions of the nerve at the base* of the brain may result from the pressure of tumors, meningitis (particularly the cerebro-spinal form), hæmorrhage, or traumatism. A primary degeneration of the nerve may occur in locomotor ataxia. Nuclear disease is rare. By far the most interesting form results from epidemic cerebro-spinal meningitis, in which the

nerve is frequently involved, causing permanent deafness. In young children the condition results in deaf-mutism.

(*c*) In a majority of the cases associated with auditory-nerve symptoms the lesion is in the *labyrinth*, either primary or the result of extension of disease of the middle ear. Three groups of symptoms may be produced—hyperæsthesia and irritation, diminished function or nervous deafness, and vertigo.

(1) *Hyperæsthesia and Irritation.*—This may be due to altered function of the centre as well as of the nerve ending. True hyperæsthesia—hyperacusis—is a condition in which sounds, sometimes even those inaudible to other persons, are heard with great intensity. It occurs in hysteria and occasionally in cerebral disease. As already mentioned, in paralysis of the stapedius low notes may be heard with intensity. In dysæsthesia, or dysacusis, ordinary sounds cause an unpleasant sensation, as commonly happens in connection with headache, when ordinary noises are badly borne.

Tinnitus aurium is a term employed to designate certain subjective sensations of ringing, roaring, ticking, and whirring noises in the ear. It is a very common and often a distressing symptom. It is associated with many forms of ear disease and may result from pressure of wax on the drum. It is rare in organic disease of the central connections of the nerve. Sudden intense stimulation of the nerve may cause it. A form not uncommonly met with in medical practice is that in which the patient hears a continual *bruit* in the ear, and the noise has a systolic intensification, usually on one side. I have twice been consulted by physicians for this condition under the belief that they had an internal aneurism. It occurs in conditions of anæmia and neurasthenia. Subjective noises in the ear may precede an epileptic seizure and are sometimes present in migraine. In whatever form tinnitus exists, though slight and often regarded as trivial, it occasions great annoyance and often mental distress, and has even driven patients to suicide.

The *diagnosis* is readily made; but it is often extremely difficult to determine upon what condition the tinnitus depends. The relief of constitutional states, such as anæmia, neurasthenia, or gout, may result in cure. A careful local examination of the ear should always be made. One of the most worrying forms is the constant clicking, sometimes audible many feet away from the patient, and due probably to clonic spasm of the muscles connected with the Eustachian tube or of the levator palati. The condition may persist for years unchanged, and then disappear suddenly. The pulsating forms of tinnitus, in which the sound is like that of a systolic *bruit*, are almost invariably subjective, and nothing is audible with the stethoscope. It is to be remembered that in children there is a systolic brain murmur, best heard over the ear, and in some instances is heard in the adult.

(2) *Diminished Function or Nervous Deafness.*—In testing for nervous

deafness, if the tuning-fork cannot be heard when placed near the meatus, but the vibrations are audible by placing the foot of the tuning-fork against the temporal bone, the conclusion may be drawn that the deafness is not due to involvement of the nerve. The vibrations are conveyed through the temporal bone to the cochlea and vestibule. The watch may be used for the same purpose, and if the meatus is closed and the watch is heard better in contact with the mastoid process than when opposite the open meatus, the deafness is probably not nervous. Practically, disturbance of the function of the auditory nerve is not a very frequent symptom in brain-disease, but in all cases the function of the nerve should be carefully tested

(3) **Auditory Vertigo—Menière's Disease.**—In 1861 Menière, a French physician, described an affection characterized by noises in the ear, vertigo (which might be associated with loss of consciousness), vomiting, and, in many cases, progressive loss of hearing. The term is now used to include all cases of sudden vertigo accompanied by noises in the ear and deafness. The frequency of vertigo with ear symptoms is striking. Thus, of 106 cases noted by Gowers, in which there was definite vertigo, in 94 ear symptoms were present, either tinnitus or deafness or both.

Symptoms.—The attack usually sets in suddenly with a buzzing noise in the ears and the patient feels as if he was reeling or staggering. He may feel himself to be reeling, or the objects about him may seem to be turning, or the phenomena may be combined. The attack is often so abrupt that the patient falls, though, as a rule, he has time to steady himself by grasping some neighboring object. There may be slight but transient loss of consciousness. In a few minutes, or even less, the vertigo passes off and the patient becomes pale and nauseated, a clammy sweat breaks out on the face, and vomiting may follow.

The deafness, which is always of a nervous character, may be in only one ear and is never complete. The tinnitus is described as either a roaring or a throbbing sound. Ocular symptoms may be present; thus, jerking of the eyeballs or nystagmus may develop during the attack, or diplopia.

Labyrinthine vertigo is paroxysmal, coming on at irregular intervals. Sometimes weeks or months may elapse between the attacks; in other cases there may be several attacks in a day. As a rule, the patients have no affection of the middle ear. The disease rarely occurs in young persons, is most frequent after the fortieth year, and is more common in men than in women.

The pathology of the disease has been much discussed. There are two theories concerning its origin—one, that it is due to affection of the labyrinth itself, which causes a disturbance of equilibrium, such as is proved by experiment to be associated with lesion of the semicircular canals; the other that it is really a trouble involving the centres presiding over hearing and equilibration.

It has also been held to be a vaso-motor neurosis of the vessels of the labyrinth. The condition of the labyrinth in these cases is variable. Acute disease with hæmorrhage has been described, or slow progressive degeneration of the nerves. Giddiness and vomiting may, however, be produced by irritation in other parts of the ear; thus, there are instances in which pressure on the drum or irritation of the external meatus is followed by an attack of giddiness and vomiting.

Diagnosis.—The combination of tinnitus with giddiness, with or without gastric disturbance, is sufficient to establish a diagnosis. There are other forms of vertigo from which it must be distinguished. The form known as gastric vertigo, which is associated with dyspepsia and occurs most commonly in persons of middle age, is, as a rule, readily distinguished by the absence of tinnitus or evidences of disturbance in the function of the auditory nerve. This variety of vertigo is much less common than Trousseau's description would lead us to believe.

The cardio-vascular vertigo, one of the most common forms, occurs in cases of valvular disease, particularly aortic insufficiency, and as frequently in arterio-sclerosis.

There is a remarkable form of vertigo described by Gerlier, which is characterized by attacks of paretic weakness of the extremities, falling of the eyelids, remarkable depression, but with retention of consciousness. It attacks only men, and has occurred in epidemic form among laborers in the canton of Geneva.

Aural vertigo must be carefully distinguished from attacks of *petit mal*, or, indeed, of definite epilepsy. It is rare in *petit mal* to have noises in the ear or actual giddiness, but in the aura preceding an epileptic attack the patient may feel giddy. Giddiness and transient loss of consciousness may be associated with organic disease of the brain, more particularly with tumor. Vomiting also may be present. A careful investigation of the symptoms will usually lead to a correct diagnosis.

The outlook in Menière's disease is uncertain. While many cases recover completely, in others deafness results and the attacks recur at shorter intervals. In aggravated cases the patient constantly suffers from vertigo and may even be confined to his bed.

Treatment.—Bromide of potassium, in twenty-grain doses three times a day, is sometimes beneficial. If there is a history of syphilis, the iodide should be administered. The salicylates are recommended, and Charcot advises quinine to cinchonism. In cases in which there is increase in the arterial tension nitroglycerine may be given, at first in very small doses, but increasing gradually. It is not specially valuable in Menière's disease, but in the cases of giddiness in middle-aged men and women associated with arterio-sclerosis it sometimes acts very satisfactorily.

VII. Glosso-pharyngeal Nerve.

This nerve contains both motor and sensory fibres and is also a nerve of the special sense of taste to the tongue. It supplies, by its motor branches, the stylo-pharyngeus and the middle constrictor of the pharynx. The sensory fibres are distributed to the upper part of the pharynx.

Symptoms.—Of nuclear disturbance we know very little. The pharyngeal symptoms of bulbar paralysis are probably associated with involvement of the nuclei of this nerve. Lesion of the nerve trunk itself is rare, but it may be compressed by tumors or involved in meningitis. Disturbance of the sense of taste may result from loss of function of this nerve, in which case it is chiefly in the posterior part of the tongue and soft palate. Gowers, however, states that there is no case on record in which loss of taste in these regions has been produced by disease of the roots of the glosso-pharyngeal; whereas, on the other hand, disease of the root of the fifth nerve may cause loss of taste on the back as well as the front of the tongue, as if the taste fibres of the glosso-pharyngeal came from the fifth.

The general disturbances of the sense of taste may here be briefly referred to. Loss of the sense of taste—*ageusia*—may be caused by disturbance of the peripheral end organs, as in affections of the mucosa of the tongue. This is very common in the dry tongue of fever or the furred tongue of dyspepsia, under which circumstances, as the saying is, everything tastes alike. Strong irritants too, such as pepper, tobacco, or vinegar, may dull or diminish the sense of taste. Complete loss may be due to involvement of the nerves either in their course or in the centres. Disturbance in the sense of taste is most commonly seen in involvement of the fifth nerve, and it may be that this nerve alone subserves the function. Perversion of the sense of taste—*parageusis*—is rarely found, except as an hysterical manifestation and in the insane. Increased sensitiveness is still more rare. There are occasional subjective sensations of taste, occurring as an aura in epilepsy or as part of the hallucinations in the insane.

To test the sense of taste the patient's eyes should be closed and small quantities of various substances applied. The sensation should be perceived before the tongue is withdrawn. The following are the most suitable tests: For bitter, quinine; for sweetness, a strong solution of sugar or saccharin; for acidity, vinegar; and for the saline test, common salt. One of the most important tests is the feeble galvanic current, which gives the well-known metallic taste.

VIII. Pneumogastric Nerve.

The vagus nerve has an important and extensive distribution, supplying the pharynx, larynx, lungs, heart, œsophagus, and stomach. The nerve may be involved at its nucleus with the spinal accessory and the hypoglossal, forming what is known as bulbar paralysis. It may be com-

pressed by tumors or aneurism, or in the exudation of meningitis, simple or syphilitic. In its course in the neck the trunk may be involved by tumors or in wounds. It has been tied in ligature of the carotid, and has been cut in the removal of deep-seated tumors. The trunk may be attacked by neuritis.

The affections of the vagus are best considered in connection with the distribution of the separate nerves.

(*a*) **Pharyngeal Branches.**—In combination with the glosso-pharyngeal the branches from the vagus form the pharyngeal plexus, from which the muscles and mucosa of the pharynx are supplied. In *paralysis* due to involvement of this either in the nuclei, as in bulbar paralysis, or in the course of the nerve, as in diphtheritic neuritis, there is difficulty in swallowing and the food is not passed on into the œsophagus. If the nerve on one side only is involved, the deglutition is not much impaired. In these cases the particles of food frequently pass into the larynx, and, when the soft palate is involved, into the posterior nares.

Spasm of the pharynx is always a functional disorder, usually occurring in hysterical and nervous people. Gowers mentions a case of a gentleman who could not eat unless alone, on account of the inability to swallow in the presence of others from spasm of the pharynx. This spasm is a well-marked feature in hydrophobia, and I have seen it in a case of pseudo-hydrophobia.

(*b*) **Laryngeal Branches.**—The superior laryngeal nerve supplies the mucous membrane of the larynx above the cords and the crico-thyroid muscle. The inferior or recurrent laryngeal curves around the arch of the aorta on the left side and the subclavian artery on the right, passes along the trachea and supplies the mucosa below the cords and all the muscles of the larynx except the crico-thyroid and the epiglottidean. Experiments have shown that these motor nerves of the pneumogastric are all derived from the spinal accessory. The remarkable course of the recurrent laryngeal nerves renders them liable to pressure by tumors within the thorax, particularly by aneurism. The following are the most important forms of paralysis:

(1) *Bilateral Paralysis of the Abductors.*—In this condition, the posterior crico-arytenoids are involved and the glottis is not opened during inspiration. The cords may be close together in the position of phonation, and during inspiration may be brought even nearer together by the pressure of air, so that there is only a narrow chink through which the air whistles with a noisy stridor. This dangerous form of laryngeal paralysis occurs occasionally as a result of cold, or may follow a laryngeal catarrh. The posterior muscles have been found degenerated when the others were healthy. The condition may be produced by pressure upon both vagi, or upon both recurrent nerves. As a central affection it occurs in tabes and bulbar paralysis, but may occur also in hysteria. The characteristic symptoms are inspiratory stridor with unimpaired phonation. Possibly,

as Gowers suggests, many cases of so-called hysterical spasm of the glottis are in reality abductor paralysis.

(2) *Unilateral Abductor Paralysis.*—This frequently results from the pressure of tumors or involvement of one recurrent nerve. Aneurism is by far the most common cause, though on the right side the nerve may be involved in thickening of the pleura. The symptoms are hoarseness or roughness of the voice, such as is so common in aneurism. Dyspnœa is not often present. The cord on the affected side does not move in inspiration. Subsequently the adductors may also become involved, in which case the phonation is still more impaired.

(3) *Adductor Paralysis.*—This results from involvement of the lateral crico-arytenoid and the arytenoid muscle itself. It is common in hysteria, particularly of women, and causes the hysterical aphonia, which may come on suddenly. It may result from catarrh of the larynx or from overuse of the voice. In laryngoscopic examination it is seen, on attempt at phonation, that there is no power to bring the cords together. In this connection the following table from Gowers work will be found valuable to the student:

Symptoms.	Signs.	Lesion.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Spasm of the Muscles of the Larynx.—In this the adductor muscles are involved. It is not an uncommon affection in children, and has already been referred to as laryngismus stridulus. Paroxysmal attacks of laryngeal spasm are rare in the adult, but cases are described in which the patient, usually a young girl, wakes at night in an attack of intense dyspnœa, which may persist long enough to produce cyanosis. Liveing states that they may replace attacks of migraine. They occur in a characteristic form in locomotor ataxia, forming the so-called laryngeal crises. There is a condition known as spastic aphonia, in which, when the patient attempts to speak, phonation is completely prevented by a spasm.

Disturbance of the sensory nerves of the larynx is rare.

Anæsthesia may occur in bulbar paralysis and in diphtheritic neuritis—a serious condition, as portions of food may enter the windpipe. It is usually associated with dysphagia and is sometimes present in hysteria. Hyperæsthesia of the larynx is rare.

(*c*) **Cardiac Branches.**—The cardiac plexus is formed by the union of branches of the vagi and of the sympathetic nerves. The vagus fibres subserve motor, sensory, and probably trophic functions.

(1) *Motor.*—The fibres which inhibit, control, and regulate the cardiac action pass in the vagi. Irritation may produce slowing of the action. Czermak could slow or even arrest the heart's action for a few beats by pressing a small tumor in his neck against one pneumogastric nerve, and it is said that the same can be produced by forcible bilateral pressure on the carotid canal. There are instances in which persons appear to have had voluntary control over the action of the heart. The most remarkable instance was that of Colonel Townsend, who could slow the action of the heart at will. Retardation of the heart's action has also followed accidental ligature of one vagus. Irritation at the nuclei may also be accompanied by extreme slowness. The condition of brachycardia may be associated with a neurosis of this nerve. On the other hand, when there is complete paralysis of the vagi, the inhibitory action may be abolished and the acceleratory influences have full sway. The heart's action is then greatly increased. This is seen in some instances of diptheritic neuritis and in involvement of the nerve by tumors, or its accidental removal or ligature. Complete loss of function of one vagus may, however, not be followed by any symptoms.

(2) *Sensory* symptoms on the part of the cardiac branches are very varied. Normally, the heart's action proceeds regularly without the participation of consciousness, but the unpleasant feelings and sensations of palpitation and pain are conveyed to the brain through this nerve. How far the fibres of the pneumogastric are involved in angina it is impossible to say. The various disturbances of sensation are described under the cardiac neuroses.

(*d*) **Pulmonary Branches.**—We know very little of the pulmonary branches of the vagi. The motor fibres are stated to control the action of

the bronchial muscles, and it has long been held that asthma may be a neurosis of these fibres. The various alterations in the respiratory rhythm are probably due more to changes in the centre than in the nerves themselves.

(*e*) **Gastric and Œsophageal Branches.**—The muscular movements of these parts are presided over by the vagi and vomiting is induced through them, usually reflexly, but also by direct irritation, as in meningitis. Spasm of the œsophagus generally occurs with other nervous phenomena. Gastralgia may sometimes be due to cramp of the stomach, but is more commonly a sensory disturbance of this nerve, due to direct irritation of the peripheral ends, or is a neuralgia of the terminal fibres. Hunger is said to be a sensation aroused by the pneumogastric, and some forms of nervous dyspepsia probably depend upon disturbed function of this nerve. The severe gastric crises which occur in locomotor ataxia are due to central irritation of the nuclei. Some describe exophthalmic goitre under lesions of the vagi.

IX. Spinal Accessory Nerve.

Paralysis.—The smaller or internal part of this nerve joins the vagus and is distributed through it to the laryngeal muscles. The larger external part is distributed to the sterno-mastoid and trapezius muscles.

The nuclei of the nerve, particularly of the accessory part, may be involved in bulbar paralysis. The nuclei of the external portion, situated as they are in the cervical cord, may be attacked in progressive degeneration of the motor nuclei of the cord. The nerve may be involved in the exudation of meningitis, or be compressed by tumors, or in caries. The *symptoms* of paralysis of the accessory portion which joins the vagus have already been given in the account of the palsy of the laryngeal branches of the pneumogastric. Disease or compression of the external portion is followed by paralysis of the sterno-mastoid and of the trapezius on the same side. In paralysis of one sterno-mastoid, the patient rotates the head with difficulty to the opposite side, but there is no torticollis, though in some cases the head is held obliquely. As the trapezius is supplied in part from the cervical nerves, it is not completely paralyzed, but the portion which passes from the occipital bone to the acromion is functionless. The paralysis of the muscle is well seen when the patient draws a deep breath or shrugs the shoulders. The middle portion of the trapezius is also weakened, the shoulder droops a little, and the angle of the scapula is rotated inward by the action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is impaired, for the trapezius does not fix the scapula as a point from which the deltoid can work.

In progressive muscular atrophy we sometimes see bilateral paralysis of these muscles. Thus, if the sterno-mastoids are affected, the head tends to fall back; when the trapezii are involved, it falls forward, a characteristic attitude of the head in many cases of progressive muscular

atrophy. Gowers suggests that lesions of the accessory in difficult labor may account for those cases in which during the first year of life the child has great difficulty in holding up the head. In children this drooping of the head is an important symptom in cervical meningitis, the result of caries.

The *treatment* of the condition depends much upon the cause. In the central nuclear atrophy but little can be done. In paralysis from pressure the symptoms may gradually be relieved. The paralyzed muscles should be stimulated by electricity and massage.

Accessory Spasm.—(*Torticollis; Wryneck.*)—The forms of spasm affecting the cervical muscles are best considered here, as the muscles supplied by the accessory are chiefly, though not solely, responsible for the condition. The following forms may be described in this section:

(*a*) *Congenital Torticollis.*—This condition, also known as fixed torticollis, depends upon the shortening and atrophy of the sterno-mastoid on one side. It occurs in children and may not be noticed for several years on account of the shortness of the neck, the parents often alleging that it has only recently come on. It affects the right side almost exclusively. A remarkable circumstance in connection with it is the existence of facial asymmetry noted by Wilks, which appears to be an essential part of this congenital form. It occurred in six cases reported by Golding-Bird. In a case recently under my observation, the wryneck was not noticed until her tenth year. The muscle was divided and she seemed quite well; but as she developed the asymmetry of the face became very striking. In congenital wryneck the sterno-mastoid is shortened, hard and firm, and in a condition of more or less advanced atrophy. This must be distinguished from the local thickening in the sterno-mastoid due to rupture, which may occur at the time of birth and produce an induration or muscle callus. Although the sterno-mastoid is almost always affected, there are rare cases in which the fibrous atrophy affects the trapezius. This form of wryneck in itself is unimportant, since it is readily relieved by tenotomy, but Golding-Bird states that the facial asymmetry persists, or indeed may, as shown by photographs in my case, become more evident. With reference to the pathology of the affection, Golding-Bird concludes that the facial asymmetry and the torticollis are integral parts of one affection which has a central origin and is the counterpart in the head and neck of infantile paralysis with talipes in the foot.

(*b*) *Spasmodic Wryneck.*—Two varieties of this spasm occur, the tonic and the clonic, which may alternate in the same case; or, as is most common, they are separate and remain so from the outset. The disease is most frequent in adults and, according to Gowers, most common in females. In this country it is certainly more frequent in males. Of the eight or ten cases which came under my observation in Montreal and Philadelphia, all were males. In females it may be an hysterical manifestation. There may be a marked neurotic family history, but it is usually

impossible to fix upon any definite etiological factor. Some cases have followed cold; others a blow.

The *symptoms* are well defined. In the tonic form the contracted sterno-mastoid draws the occiput toward the shoulder of the affected side; the chin is raised, and the face rotated to the other shoulder. The sterno-mastoid may be affected alone or in association with the trapezius. When the latter is implicated the head is depressed still more toward the same side. In long-standing cases these muscles are prominent and very rigid. There may be some curvature of the spine, the convexity of which is toward the sound side. The cases in which the spasm is clonic are much more distressing and serious. The spasm is rarely limited to a single muscle. The sterno-mastoid is almost always involved and rotates the head so as to approximate the mastoid process to the inner end of the clavicle, turning the face to the opposite side and raising the chin. When with this the trapezius is affected, the depression of the head toward the same side is more marked. The head is drawn somewhat backward; the shoulder, too, is raised by its action. According to Gowers, the splenius is associated with the sterno-mastoid about half as frequently as the trapezius. Its action is to incline the head and rotate it slightly toward the same side. Other muscles may be involved, such as the scalenus and platysma myoides; and in rare cases the head may be rotated by the deep cervical muscles, the rectus and obliquus. There are cases in which the spasm is bilateral, causing a backward movement—the retro-collic spasm. This may be either tonic or clonic, and in extreme cases the face is horizontal and looks upward.

These clonic contractions may come on without warning, or be preceded for a time by irregular pains or stiffness of the neck. The jerking movements recur every few moments, and it is impossible to keep the head still for more than a minute or two. In time the muscles undergo hypertrophy and may be distinctly larger on one side than the other. In some cases the pain is considerable; in others there is simply a feeling of fatigue. The spasms cease during sleep. Emotion, excitement, and fatigue increase them. The spasm may extend from the muscles of the neck and involve those of the face or of the arms.

The disease varies much in its course. Cases occasionally get well, but the great majority of them persist, and, even if temporarily relieved, the disease frequently recurs. The affection is usually regarded as a functional neurosis, but it is possibly due to disturbance of the cortical centres presiding over the muscles.

Treatment.—Temporary relief is sometimes obtained; a permanent cure is exceptional. Various drugs have been used, but rarely with benefit. Occasionally, large doses of bromide will lessen the intensity of the spasm. Morphia, subcutaneously, has been successful in some reported cases, but there is the great danger of establishing the morphia habit. Galvanism may be tried. Counter-irritation is probably

useless. Fixation of the head mechanically can rarely be borne by the patient. These obstinate cases fall ultimately into the hands of the surgeon, and the operations of stretching, division, and excision of the accessory nerve and division of the muscles have been tried. The latter does not check the spasm, and may aggravate the symptoms. Temporary relief may follow, but, as a rule, the condition returns. In the cases of spasm of the deep-seated muscles, Keen has devised an operation for their section.

(*c*) The *nodding spasm* of children may here be mentioned as involving chiefly the muscles innervated by the accessory nerve. It may be a simple trick, a form of habit spasm, or a phenomenon of epilepsy (E. nutans), in which case it is associated with transient loss of consciousness. A similar nodding spasm may occur in older children. In women it sometimes occurs as an hysterical manifestation, commonly as part of the so-called salaam convulsion.

X. Hypoglossal Nerve.

This is the motor nerve of the tongue and for most of the muscles attached to the hyoid bone. Its cortical centre is probably the lower part of the ascending frontal gyrus.

Paralysis.—(1) *Central Lesion.*—The tongue is often paralyzed in hemiplegia, and the paralysis may result from a lesion of the cortex itself, or of the fibres as they pass to the medulla. It does not occur alone and will be considered with hemiplegia. There is this difference, however, between the cortical and other forms, that the muscles on both sides of the tongue may be more or less affected but do not waste, nor are their electrical reactions disturbed.

(2) *Nuclear* and *infra-nuclear* lesions of the hypoglossal result from slow progressive degeneration, as in bulbar paralysis or in locomotor ataxia, and occasionally there is acute softening from obstruction of the vessels. Trauma and lead poisoning have also been assigned as causes. The fibres may be damaged by a tumor, and at the base by meningitis; or the nerve is sometimes involved in its foramen by disease of the skull. The nuclei of both nerves are usually affected together, but may be attacked separately. As a result, there is loss of function in the nerve fibres and the tongue undergoes atrophy on the affected side. It is protruded toward the paralyzed side and may show fibrillary twitching.

The *symptoms* of involvement of one hypoglossal, either at its centre or in its course, are those of unilateral paralysis and atrophy of the tongue. When protruded, it is pushed toward the affected side, and there are fibrillary twitchings. The atrophy is usually marked and the mucous membrane on the affected side is thrown into folds. Articulation is not much impaired in the unilateral affection. When the disease is bilateral, the tongue lies almost motionless in the floor of the mouth; it is atrophied,

and cannot be protruded. Speech and mastication are extremely difficult and deglutition may be impaired. If the seat of the disease is above the nuclei, there may be little or no wasting. The condition is seen in progressive bulbar paralysis and occasionally in progressive muscular atrophy.

The *diagnosis* is readily made and the situation of the lesion can usually be determined, since when supra-nuclear there is associated hemiplegia and no wasting of the muscles of the tongue. Nuclear disease is only occasionally unilateral; most commonly bilateral and part of a bulbar paralysis. It should be borne in mind that the fibres of the hypoglossal may be involved within the medulla after leaving their nuclei. In such a case there may be paralysis of the tongue on one side and paralysis of the limbs on the opposite side, and the tongue, when protruded, is pushed toward the sound side.

Spasm.—This rare affection may be unilateral or bilateral. It is most frequently a part of some other convulsive disorder, such as epilepsy, chorea, or spasm of the facial muscles. In some cases of stuttering, spasm of the tongue precedes the explosive utterance of the words. It may occur in hysteria, and is said to follow reflex irritation in the fifth nerve. The most remarkable cases are those of paroxysmal clonic spasm, in which the tongue is rapidly thrust in and out, as many as forty or fifty times a minute. In the case reported by Gowers the attacks occurred during sleep and continued for a year and a half. The spasm is usually bilateral. Wendt has reported a case in which it was unilateral. The prognosis is usually good.

IV. DISEASES OF THE SPINAL NERVES.

Cervical Plexus.

(1) **Occipito-cervical Neuralgia.**—This involves the nerve territory supplied by the second, the occipitalis major and minor, and the auricularis magnus nerves. The pains are chiefly in the back of the head and neck and in the ear. The condition may follow cold and is sometimes associated with stiffness of the neck or torticollis. Unless connected with disease of the bones or due to pressure of tumors, the outlook is usually good. There are tender points midway between the mastoid process and the spine and just above the parietal eminence, and between the sternomastoid and the trapezius. The affection may be due to direct pressure, in persons who carry very heavy loads on the neck.

(2) **Affections of the Phrenic Nerve.**—Paralysis may follow a lesion in the anterior horns at the level of the third and fourth cervical nerves, or may be due to compression of the nerve by tumors or aneurism. More rarely paralysis results from neuritis.

It may be part of a diphtheritic or lead palsy and is usually bilateral.

When the diaphragm is paralyzed respiration is carried on by the intercostal and accessory muscles. When the patient is quiet and at rest little may be noticed, but the abdomen retracts in inspiration and is forced out in expiration. On exertion or even on attempting to move there may be dyspnœa. If the paralysis sets in suddenly there may be dypnœa and lividity, which is usually temporary (W. Pasteur). Intercurrent attacks of bronchitis seriously aggravate the condition. Difficulty in coughing, owing to the impossibility of drawing a full breath, adds greatly to the danger of this complication, as the mucus accumulates in the tubes.

When the phrenic nerve is paralyzed on one side the paralysis may be scarcely noticeable, but careful inspection shows that the descent of the diaphragm is much less on the affected side.

The *diagnosis* of paralysis is not always easy, particularly in women, who habitually use this muscle less than men, and in whom the diaphragmatic breathing is less conspicuous. Immobility of the diaphragm is not uncommon, particularly in diaphragmatic pleurisy, in large effusions, and in extensive emphysema. The muscle itself may be degenerated and its power impaired.

Owing to the lessened action of the diaphragm, there is a tendency to accumulation of blood at the bases of the lungs, and there may be impaired resonance and signs of œdema. As a rule, however, the paralysis is not confined to this muscle, but is part of a general neuritis or an anterior polio-myelitis, and there are other symptoms of value in determining its presence. The outlook is usually serious. Pasteur states that of fifteen cases following diphtheria, only eight recovered. The treatment is that of the neuritis or polio-myelitis with which it is associated.

Brachial Plexus.

(1) **Combined Paralysis.**—The plexus may be involved in the supraclavicular region by compression of the nerve trunks as they leave the spine, or by tumors and other morbid processes in the neck. Below the clavicle lesions are more common and result from injuries following dislocation or fracture, sometimes from neuritis. The most common cause of lesion of the brachial plexus is luxation of the humerus, particularly the subcoracoid form. If the dislocation is quickly reduced the symptoms are quite transient, and disappear in a few days. In severe cases all the branches of the plexus, or only one or two, may be involved. The most serious cases are those in which the dislocation is undetected or unreduced for some time, when the prolonged pressure on the nerves may cause complete and permanent paralysis of the arm. The muscles waste, the reaction of degeneration is present, and trophic changes in the skin are apt to occur. The medico-legal bearings of these cases are important, and may be thus briefly summarized: Direct injury, as by a fall or blow on the shoulder, resulting in great bruising of the nerves without dislocation, is

occasionally followed by complete paralysis of the arm. A dislocation may be set immediately and yet the lesion of the brachial plexus may be such as to cause permanent paralysis of the nerves. The dislocation may be reduced and the joint in subsequent movements slips out again. It has happened that by the time the surgeon sees the patient again, the damage has become irreparable.

Injuries and blows on the neck may cause partial paralysis of the arm, involving the deltoid, supraspinatus, infraspinatus, biceps, brachialis anticus, and the supinator. The injury may occur to the child during delivery.

A primary neuritis of the brachial plexus is rare. More commonly the process is an ascending neuritis from a lesion of a peripheral branch, involving first the radial or ulnar nerves, and spreading upward to the plexus, producing gradually complete loss of power in the arm.

(2) **Lesions of Individual Nerves of the Plexus.**—(*a*) *Long Thoracic Nerve* (*Serratus Palsy*).—This occurs chiefly in men. The nerve is injured in the posterior triangle of the neck, usually by direct pressure in the carrying of loads; cold may cause neuritis. It may be involved also in progressive muscular atrophy and in polio-myelitis anterior. When paralyzed the scapula on the affected side looks winged, which results from the projection of the angle and posterior border. This is particularly noticeable when the arm is moved forward, when the serratus no longer holds the scapula against the thorax. It is a well-defined and readily recognized form of paralysis. The onset is associated with, sometimes preceded by, neuralgic pains. The course is dubious, and many months may elapse before there is any improvement.

(*b*) *Circumflex Nerve.*—This supplies the deltoid and the teres minor. The nerve is apt to be involved in injuries, in dislocations, bruising by a crutch, or sometimes by extension of inflammation from the joint. Occasionally the paralysis arises from a pressure neuritis during an illness. As a consequence of loss of power in the deltoid, the arm cannot be raised. The wasting is usually marked and changes the shape of the shoulder. Sensation may also be impaired in the skin over the muscle. The joint may be relaxed and there may be a distinct space between the head of the humerus and the acromion. In other instances the ligaments are thickened, and a condition not unlike ankylosis may be produced, which is readily distinguished on moving the arm.

(*c*) *Musculo-spiral Paralysis; Radial Paralysis.*—This is one of the most common of peripheral palsies, and results from the exposed position of the musculo-spiral nerve. It is often bruised in the use of the crutch, by injuries of the arm, blows, or fractures. It is frequently injured when a person falls asleep with the arm over the back of a chair, or by pressure of the body upon the arm when a person is sleeping on a bench or on the ground. It may be paralyzed by sudden violent contraction of the triceps. It is sometimes involved in a neuritis from cold, but this is uncommon in

comparison with other causes. In the subcutaneous injection of ether the nerve may be accidentally struck and temporarily paralyzed. The paralysis of lead poisoning is the result of involvement of certain branches of this nerve.

A lesion when high up involves the triceps, the brachialis anticus, and the supinator longus, as well as the extensors of the wrist and fingers. Naturally, in lesions just above the elbow the arm muscles and the supinator longus are spared. The most characteristic feature of the paralysis is the wrist-drop and the inability to extend the first phalanges of the fingers and thumb. In the pressure palsies the supinators are usually involved and the movements of supination cannot be accomplished. The sensations may be impaired, or there may be marked tingling, but the loss of sensation is rarely so pronounced as that of motion.

The affection is readily recognized, but it is sometimes difficult to say upon what it depends. The sleep and pressure palsies are, as a rule, unilateral and involve the supinator longus. The paralysis from lead is bilateral and the supinators are unaffected. Bilateral wrist-drop is a very common symptom in many forms of multiple neuritis, particularly the alcoholic; but the mode of onset and the involvement of the legs and arms are features which make the diagnosis easy. The duration and course of the musculo-spiral paralysis are very variable. The pressure palsies may disappear in a few days. Recovery is the rule, even when the affection lasts for many weeks. The electrical examination is of importance in the prognosis, and the rules laid down under paralysis of the facial nerve hold good here.

The treatment is that of neuritis.

(*d*) *Ulnar Nerve.*—The motor branches supply the ulnar halves of the deep flexor of the fingers, the muscles of the little finger, the interossei, the adductor and the inner head of the short flexor of the thumb, and the ulnar flexor of the wrist. The sensory branches supply the ulnar side of the hand—two and a half fingers on the back, and one and a half fingers on the front. Paralysis may result from pressure, usually at the elbow-joint, although the nerve is here protected. Possibly the neuritis in the ulnar nerve in some cases of acute illness may be due to this cause. Gowers mentions the case of a lady who twice had ulnar neuritis after confinement. Owing to paralysis of the ulnar flexor of the wrist, the hand moves toward the radial side; adduction of the thumb is impossible; the first phalanges cannot be flexed, and the others cannot be extended. In long-standing cases the first phalanges are overextended and the others strongly flexed, producing the claw-hand; but this is not so marked as in the progressive muscular atrophy. The loss of sensation corresponds to the sensory distribution just mentioned.

(*e*) *Median Nerve.*—This supplies the flexors of the fingers except the ulnar half of the deep flexors, the abductor and the flexors of the thumb, the two radial lumbricales, the pronators, and the radial flexor of the wrist.

The sensory fibres supply the radial side of the palm and the front of the thumb, the first two fingers and half the third finger, and the dorsal surfaces of the same three fingers.

This nerve is seldom involved alone. Paralysis results from injury and occasionally from neuritis. The signs are inability to pronate the forearm beyond the mid-position. The wrist can only be flexed toward the ulnar side; the thumb cannot be opposed to the tips of fingers. The second phalanges cannot be flexed on the first; the distal phalanges of the first and second fingers cannot be flexed; but in the third and fourth fingers this action can be performed by the ulnar half of the flexor profundus. The loss of sensation is in the region corresponding to the sensory distribution already mentioned. The wasting of the thumb muscles, which is usually marked in this paralysis, gives to it a characteristic appearance.

Lumbar and Sacral Plexuses.

The *lumbar plexus* is sometimes involved in growths of the lymph glands, in psoas abscess, and in disease of the bones of the vertebræ. Of its branches the *obturator nerve* is occasionally injured during parturition When paralyzed the power is lost over the adductors of the thigh and one leg cannot be crossed over the other. Outward rotation is also disturbed. The *anterior crural nerve* is sometimes involved in wounds or in dislocation of the hip-joint, less commonly during parturition, and sometimes by disease of the bones and in psoas abscess. The special symptoms of affection of this nerve are paralysis of the extensors of the knee with wasting of the muscles, anæsthesia of the antero-lateral parts of thigh and of the inner side of the leg to the big toe. This nerve is sometimes involved early in growths about the spine, and there may be pain in its area of distribution. Loss of the power of abducting the thigh results from paralysis of the *gluteal nerve*, which is distributed to the gluteus, medius, and minimus muscles.

The *sacral plexus* is frequently involved in tumors and inflammations within the pelvis and may be injured during parturition. Neuritis is common, usually an extension from the sciatic nerve.

Of the branches, the *sciatic nerve*, when injured at or near the notch, causes paralysis of the flexors of the legs and the muscles below the knee, but injury below the middle of the thigh involves only the latter muscles. There is also anæsthesia of the outer half of the leg, the sole, and the greater portion of the dorsum of the foot. Wasting of the muscles frequently follows, and there may be trophic disturbances. In paralysis of one sciatic the leg is fixed at the knee by the action of the quadriceps extensor and the patient is able to walk.

Paralysis of the *small sciatic nerve* is rarely seen. The gluteus maximus is involved and there may be difficulty in rising from a seat. There is a strip of anæsthesia along the back of the middle third of the thigh.

External Popliteal Nerve.—Paralysis involves the peronæi, the long extensor of the toes, tibialis anticus, and the extensor brevis digitorum. The ankle cannot be flexed, resulting in a condition known as foot-drop, and as the toes cannot be raised the whole leg must be lifted, producing the characteristic *steppage* gait seen in so many forms of peripheral neuritis. In long-standing cases the foot is permanently extended and there is wasting of the anterior tibial and peroneal muscles. The loss of sensation is in the outer half of the front of the leg and on the dorsum of the foot.

Internal Popliteal Nerve.—When paralyzed plantar flexion of the foot and flexion of the toes are impossible. The foot cannot be adducted, nor can the patient rise on tiptoe. In long-standing cases talipes calcaneus follows and the toes assume a claw-like position from secondary contracture, due to overextension of the proximal and flexion of the second and third phalanges.

Sciatica.

This is, as a rule, a neuritis either of the sciatic nerve or of its cords of origin. It may in some instances be a functional neurosis or neuralgia.

It occurs most commonly in adult males. A history of rheumatism or of gout is present in many cases. Exposure to cold, particularly after heavy muscular exertion, or a severe wetting are not uncommon causes. Within the pelvis the nerves may be compressed by large ovarian or uterine tumors, by lymphadenomata, by the fœtal head during labor, and occasionally lesions of the hip-joint induce a secondary sciatica. The condition of the nerve has been examined in a few cases, and it has often been seen in the operation of stretching. It is, as a rule, swollen, reddened, and in a condition of interstitial neuritis. The affection may be most intense at the sciatic notch or in the nerve about the middle of the thigh.

Of the *symptoms*, pain is the most constant and troublesome. The onset may be severe, with slight pyrexia, but, as a rule, it is gradual, and for a time there is only slight pain in the back of the thigh, particularly in certain positions or after exertion. Soon the pain becomes more intense, and instead of being limited to the upper portion of the nerve, extends down the thigh, reaching the foot and radiating over the entire distribution of the nerve. The patient can often point out the most sensitive spots, usually at the notch or in the middle of the thigh; and on pressure these are exquisitely painful. The pain is described as gnawing or burning, and is usually constant, but in some instances is paroxysmal, and often worse at night. On walking it may be very great; the knee is [illegible] and the patient treads on the toes, so as to relieve the tension on the nerve. In protracted cases there is wasting of the muscles, but the reaction of degeneration can seldom be obtained. In these chronic cases cramp may occur and fibrillar contractions. Herpes may develop, but this is un-

usual. In rare instances the neuritis ascends and involves the spinal cord.

The duration and course are extremely variable. As a rule it is an obstinate affection, lasting for months, or even, with slight remissions, for years. Relapses are not uncommon, and the disease may be relieved in one nerve only to appear in the other. In the severer forms the patient is bedridden, and such cases prove among the most distressing and trying which the physician is called upon to treat.

In the *diagnosis* it is important, in the first place, to determine whether the disease is primary, or secondary to some affection of the pelvis or of the spinal cord. A careful rectal examination should be made, and, in women, pelvic tumor should be excluded. Lumbago may be confounded with it. Affections of the hip-joint are easily distinguished by the absence of tenderness in the course of the nerve and the sense of pain on movement of the hip-joint or on pressure in the region of the trochanter. There are instances of sacro-iliac disease in which the patient complains of pain in the upper part of the thigh, which may sometimes radiate; but careful examination will readily distinguish between the affections. Pressure on the nerve trunks of the cauda equina, as a rule, causes bilateral pain and disturbances of sensation, and, as double sciatica is rare, these circumstances always suggest lesion of the nerve roots. Between the severe lightning pains of tabes and sciatica the differences are usually well defined.

Treatment.—The pelvic organs should be carefully and systematically examined. Constitutional conditions, such as rheumatism and gout, should receive appropriate treatment. In a few cases with pronounced rheumatic history, which come on acutely with fever, the salicylates seem to do good. In other instances they are quite useless. If there is a suspicion of syphilis the iodide of potassium should be employed, and in gouty cases salines.

Rest in bed with fixation of the limb by means of a long splint is a most valuable method of treatment in many cases, one upon which Weir Mitchell has specially insisted. I have known it to relieve, and in some instances to cure, obstinate and protracted cases which had resisted all other treatment. Hydrotherapy is sometimes satisfactory, particularly the warm baths or the mud baths. Many cases are relieved by a prolonged residence at one of the thermal springs.

Antipyrin, antifebrin, and quinine, are of doubtful benefit.

Local applications are more beneficial. The hot iron or the thermocautery or blisters relieve the pain temporarily. Deep injections into the nerves give great relief and may be necessary for the pain. It is best to use cocaine at first, in doses of from an eighth to a quarter of a grain. If the pain is unbearable morphia may be used, but it is a dangerous remedy in sciatica and should be withheld as long as possible. The disease is so protracted, so liable to relapse, and the patient's *morale* so undermined by

the constant worry and the sleepless nights, that the danger of contracting the morphia habit is very great. On no consideration should the patient be permitted to use the hypodermic needle himself. It is remarkable how promptly, in some cases, the injection of distilled water into the nerve will relieve the pain. Acupuncture may also be tried; the needles should be thrust deeply into the most painful spot for a distance of about two inches, and left for from fifteen to twenty minutes. The injection of chloroform into the nerve has also been recommended.

Electricity is an uncertain remedy. Sometimes it gives prompt relief; in other cases it may be used for weeks without the slightest benefit. It is most serviceable in the chronic cases in which there is wasting of the legs, and should be combined with massage. The galvanic current should be used; a flat electrode should be placed over the sciatic notch, and a smaller one used along the course of the nerve and its branches. In very obstinate cases nerve-stretching may be employed. It is sometimes successful; but in other instances the condition recurs and is as bad as ever.

II. DISEASES OF THE SPINAL CORD.

I. AFFECTIONS OF THE MENINGES.

Diseases of the Dura Mater.

Pachymeningitis.—The dura mater of the cord is separated by a loose connective tissue from the bony canal in which it lies, and an inflammation may involve either its outer or its inner aspect; hence the division into pachymeningitis externa and interna.

(*a*) *Pachymeningitis Externa.*—This is invariably a secondary inflammation and is occasionally met with in an *acute* form in caries or in syphilitic affections of the bone. Abscess may penetrate the spinal canal or the inflammation may even extend to the peridural tissue in long-standing decubitus. The symptoms are usually those of a compression myelitis.

The *chronic* form of external pachymeningitis, also a secondary affection, is much more common. It is a constant accompaniment of tuberculous disease of the spine and plays a very important part in the production of the symptoms. The affection may be confined to the part in immediate connection with the local disease, but in some cases the subdural space over six or eight vertebræ is occupied by caseous masses. The cord at the site of the curvature in Pott's disease may be compressed, with perhaps little or no involvement of the pia mater. The internal surface of the dura may be perfectly smooth, perhaps a little adherent to the arachnoid, while the external dura is thickened, rough, and covered with a cheesy substance of a variable degree of consistence. In some instances the dura is completely surrounded by this material; in others it is chiefly

on the anterior surface. We can understand the recovery in cases of compression paraplegia if we bear in mind that in large part the actual compression is produced by this material between the diseased vertebræ and the dura mater. The symptoms are those of myelitis from compression, often with signs of involvement of the nerve roots, such as will be mentioned in the next section.

(*b*) *Pachymeningitis interna*, described by Charcot and Joffroy, involves chiefly the cervical region (*P. cervicalis hypertrophica*). The interspace between the cord and the dura is occupied by a firm, concentrically arranged, fibrinous growth, which is seen to have developed within, not outside of, the dura mater. It is a condition anatomically identical with the hæmorrhagic pachymeningitis interna of the brain. The cord is usually compressed; the central canal may be dilated—hydromyelus—and there are secondary degenerations. The nerve roots are involved in the growth and are damaged and compressed. The extent is variable. It may be limited to one segment, but more commonly involves a considerable portion of the cervical enlargement. The disease is chronic, and in some cases presents a characteristic group of symptoms. There are intense neuralgic pains in the course of the nerves whose roots are involved. They are chiefly in the arms and in the cervical region, and vary greatly in intensity. There may be hyperæsthesia with numbness and tingling; atrophic changes may develop, and there may be areas of anæsthesia. Gradually motor disturbances appear; the arms become weak and the muscles atrophied, particularly in certain groups, as the flexors of the hand. The extensors, on the other hand, remain intact, so that the condition of claw-hand is gradually produced. The grade of the atrophy depends much upon the extent of involvement of the cervical nerve roots, and in many cases the atrophy of the muscles of the shoulders and arms becomes extreme. The condition is one of cervical paraplegia, with contractures, flexion of the wrist, and typical *main en griffe*. Usually before the arms are greatly atrophied there are the symptoms of what the French writers term the second stage—namely, involvement of the lower extremities and the gradual production of a spastic paraplegia, which may develop several months after the onset of the disease, and is due to secondary changes in the cord.

The disease runs a chronic course, lasting, perhaps, two or more years. In a few instances, in which symptoms pointed definitely to this condition, recovery has taken place. The disease is to be distinguished from amyotrophic lateral sclerosis, syringomyelic, and tumors. From the first it is separated by the marked severity of the initial pains in the neck and arms; from the second, by the absence of the sensory changes characteristic of syringomyelia. From certain tumors it is very difficult to distinguish, as, in fact, the fibrinous layers form a tumor around the cord.

The condition known as *hæmatoma* of the dura mater may occur at any part of the cord, or, in its slow, progressive form—pachymeningitis

hæmorrhagica interna—may be limited to the cervical region and produce the symptoms just mentioned. It is sometimes extensive, and may coexist with a similar condition of the cerebral dura. Cysts may occur filled with hæmorrhagic contents.

Diseases of the Pia Mater.

(*a*) Acute Spinal Meningitis; Leptomeningitis.

Etiology.—Spinal meningitis occurs: (1) In tuberculosis. This is perhaps the most common form in general practice and has already been considered. (2) In specific cerebro-spinal meningitis, which occurs endemically or epidemically, and has also been considered under its appropriate section. (3) As a secondary involvement in certain infectious diseases, pneumonia, small-pox, scarlet fever, and typhoid fever. This form is very rare Even in pneumonia, in which the cerebral meninges are frequently involved, the spinal meninges are seldom affected, except perhaps in the first two or three inches of the cervical region. (4) From injury or the extension of inflammation, as after operation on spina bifida. (5) There are cases in which the meningitis appears to have followed exposure to cold and wet.

Morbid Anatomy.—The affection may be diffused over the entire cord or localized to the cervical region. In the early stage the vessels of the pia mater are injected. The fluid in the pia-arachnoid space is slightly turbid. In some intense grades, on opening the dura the contour of the cord cannot be seen, as it is completely enveloped in a sero-fibrinous or purulent exudate, which here and there causes bulging of the arachnoid. Owing to the position of the body, the exudate is most abundant in the posterior part, or sinks to the lumbar region. In acute cases the pia itself does not look thickened, but in more chronic forms the membrane may be grayish and turbid. In a majority of instances, if the inflammation is intense, the exudate is seen in the anterior and posterior median fissures and the cortical portion of the cord is swollen and infiltrated, so the condition can be properly called meningomyelitis. The affection may be limited to the spinal meninges, but in a majority of instances it is a cerebro-spinal lesion.

Symptoms.—These have already been referred to in considering the two commonest varieties, the tuberculous and the epidemic. The disease often sets in with a chill and fever. Pain in the back, stiffness in the neck, pain on pressure along the vertebræ, tremor or spasm of the muscles, and disturbances of sensation are usually present. Girdle sensations are not common. The reflexes may be increased. Later, paralytic symptoms may develop, but they are uncommon, except in pure spinal meningitis.

The *diagnosis* is often difficult. In a large proportion of the cases supposed to be spinal meningitis the membranes are not inflamed. I have

already referred to the identity of the spinal symptoms in certain of the infectious diseases with those of acute leptomeningitis. In the case of a patient with high fever, marked stiffness of the back and neck muscles, or opisthotonus with rigidity and tremor of the muscles, it is not unnatural to make a positive diagnosis of spinal meningitis, but every symptom of the condition may be present without any inflammatory exudate. The truth of Stokes's dictum, already quoted (p. 25), has been brought home to me on many occasions. On the other hand, there are instances of well-marked leptomeningitis, more particularly the cerebro-spinal form, in which spinal symptoms are trifling or absent. To distinguish between the different forms of spinal meningitis is sometimes extremely difficult. A correct diagnosis is oftenest made in tuberculous cases, since here the prodromata are well defined and the symptoms indicative of involvement of the cerebral meninges well marked. There are cases in which the spinal meninges bear the brunt of the affection. I have already referred to one case in which the meningitis was thought to be due to traumatism. The coexistence of disease at the apex of the lungs or of local tuberculous lesions elsewhere is of great value.

The diagnosis of the epidemic form has already been considered.

(*b*) **Chronic Leptomeningitis.**—As a primary lesion this is extremely rare. It sometimes follows the prolonged use of alcohol. It occurs in connection with syphilis, trauma, and as a complication of various scleroses of the spinal cord, either systemic or insular.

Anatomically the condition is characterized by a thickening and turbidity of the pia, often with adhesions to the arachnoid and the dura. The membranes may be stained with blood-pigment. These alterations may occur in localized spots or over extensive areas. The nerve roots may be involved and thickened. The spinal cord itself is rarely affected, though strands of connective tissue may extend into the cortical zone, producing slight sclerosis. The opaque, white, cartilaginous plates which occur so often on the posterior surface of the spinal arachnoid and are sometimes adherent to the pia cause no symptoms and are not to be mistaken for this chronic meningitis.

The *symptoms* of this form are indefinite. Simple thickening of the meninges may produce no signs during life unless the spinal nerve roots are involved. In any case the diagnosis is somewhat doubtful. There are instances in which pain in the back, stiffness of the dorsal muscles, and pains radiating in the nerves of the trunk or in the extremities have been marked. Hyperæsthesia and skin eruptions may be present. When the cord is involved paralytic symptoms may develop. The reflexes are increased. The course is always chronic, lasting for many years.

The treatment is purely symptomatic. Recovery probably never occurs.

Hæmorrhage into the Spinal Membranes; Hæmatorrhachis.

In meningeal apoplexy, as it is called, the blood may be between the dura mater and the spinal canal—extrameningeal hæmorrhage—or within the dura mater—intrameningeal hæmorrhage.

(*a*) *Extrameningeal Hæmorrhage* occurs usually as a result of trauma. The exudation may be extensive without compression of the cord. The blood comes from the large plexuses of veins which surround the dura. The rupture of an aneurism into the spinal canal may produce extensive and rapidly fatal hæmorrhage.

(*b*) *Intrameningeal Hæmorrhage* is rather more common, but is rarely extensive from causes acting directly on the spinal meninges themselves. Scattered hæmorrhages are not unfrequent in the acute infectious fevers, and I have twice, in malignant small-pox, seen much effusion. Bleeding occurs also in death from convulsive disorders, such as epilepsy, tetanus, and strychnia poisoning. The most extensive hæmorrhages occur in cases in which the blood comes from rupture of an aneurism at the base of the brain, either of the basilar or vertebral. In several cases of this kind I have found a large amount of blood in the spinal meninges. In ventricular apoplexy the blood may pass from the fourth ventricle into the spinal meninges. There is a specimen in the medical museum of McGill College of the most extensive intraventricular hæmorrhage, in which the blood passed into the fourth ventricle, and descended beneath the spinal arachnoid for a considerable distance. On the other hand, hæmorrhage into the spinal meninges may possibly ascend into the brain.

The *symptoms* in moderate grades may be slight and indefinite. In the non-traumatic cases the hæmorrhage may either come on suddenly or after a day or two of uneasy sensations along the spine. As a rule, the onset is abrupt, with sharp pain in the back and symptoms of irritation in the course of the nerves. There may be muscular spasms, or paralysis may come on suddenly, either in the legs alone or both in the legs and arms. In some instances the paralysis develops more slowly and is not complete. There is no loss of consciousness, and there are no signs of cerebral disturbance. The clinical picture naturally varies with the site of the hæmorrhage. If in the lumbar region, the legs alone are involved, the reflexes may be abolished, and the action of the bladder and rectum are impaired. In the dorsal region there is more or less complete paraplegia, the reflexes are usually retained, and there are signs of disturbance in the thoracic nerves, such as girdle sensations, pains, and sometimes eruption of herpes. In the cervical region the arms as well as the legs may be involved; there may be difficulty in breathing, stiffness of the muscles of the neck, and occasionally pupillary symptoms.

The prognosis depends much upon the cause of the hæmorrhage. Recovery may take place in the traumatic cases, and in those associated with the infectious diseases.

II. AFFECTIONS OF THE BLOOD-VESSELS.

(*a*) **Congestion.**—Apart from actual myelitis, we rarely see post mortem evidences of congestion of the spinal cord, and when we do it is usually limited either to the gray matter or to a definite portion of the organ. There is necessarily, from the posture of the body post mortem, a greater degree of vascularity in the posterior portion of the cord. The white matter is rarely found congested, even when inflamed; in fact, it is remarkable how uniformly pale this portion of the cord is. The gray matter often has a reddish-pink tint, but rarely a deep reddish hue, except when myelitis is present. If we know little anatomically of conditions of congestion of the cord, we know less clinically, for there are no features in any way characteristic of it.

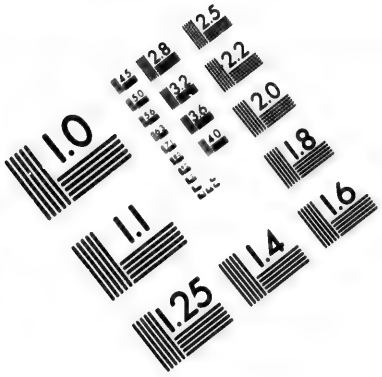
(*b*) **Anæmia.**—So, too, with this state. There may be extreme grades of anæmia of the cord without symptoms. In chlorosis and pernicious anæmia there are rarely symptoms pointing to the cord, and there is no reason to suppose that such sensations as heaviness in the limbs and tingling are especially associated with anæmia.

There are, however, some very interesting facts with reference to the profound anæmia of the cord which follows ligature of the aorta. In experiments made in Welch's laboratory by Herter, it was found that within a few moments after the application of the ligature to the aorta paraplegia came on. Paralysis of the sphincters developed, but less rapidly. Within fourteen days contractures of the limbs set in with atrophy and fibrillar twitchings. Histologically it was shown that within thirty-six hours there were marked changes in the ganglion cells of the anterior horns in the lumbar segments, and later there were signs of a definite myelitis. This condition is of interest in connection with the fact of the rapid development of a paraplegia after profuse hæmorrhage, usually from the stomach or uterus. It may come on at once or at the end of a week or ten days, and is probably due to an anatomical change in the nerve elements similar to that produced in Herter's experiments.

In this connection may be mentioned the interesting observations of Lichtheim upon the degeneration of the posterior columns of the cord in pernicious anæmia, of which he has reported three cases. He regards it as a form of toxic myelitis, due to the altered condition of the blood.

(*c*) **Embolism and Thrombosis.**—Blocking of the spinal arteries by emboli rarely occurs. It may be produced experimentally, and Money found that it was associated with choreiform movements. Thrombosis of the smaller vessels in connection with endarteritis plays an important part in many of the acute and chronic changes in the cord.

(*d*) **Endarteritis.**—It is remarkable how frequently in persons over fifty the arteries of the spinal cord are found sclerotic. The following forms may be met with: (1) A nodular peri-arteritis or endarteritis associated



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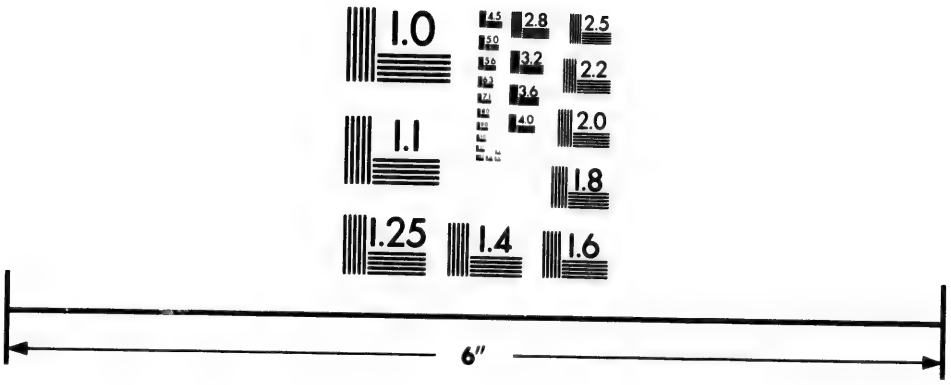

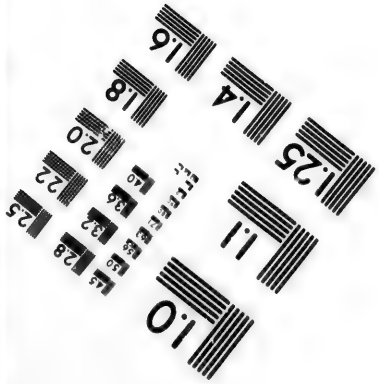

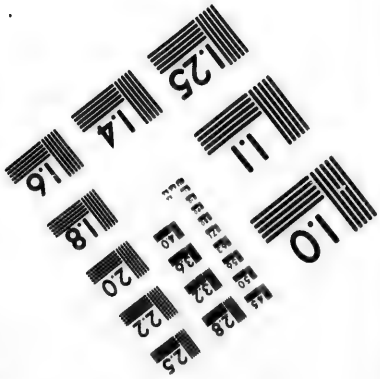

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with syphilis and sometimes with gummata of the meninges; (2) an arteritis obliterans, with great thickening of the intima and narrowing of the lumen of the vessels, involving chiefly the medium and larger-sized arteries. Miliary aneurisms or aneurisms of the larger vessels are rarely found in the spinal cord. In the classical work of Leyden but a single instance of the latter is mentioned.

(*e*) **Hæmorrhage into the Spinal Cord** (*Hæmatomyelia*).—The existence of a primary hæmorrhage into the cord has been denied on the ground that in all instances it is preceded by a condition of softening. A majority of authors, however, admit the existence of a primary form. About forty-two cases are on record, which are collected in the thesis of Hayem * and in the article of Berkeley.† It is more common in males than in females, and at the middle period of life. The cases have followed either cold and exposure or overexertion, and, most frequently of all, traumatism. It occurs also in tetanus and convulsions. Hæmorrhage may be associated with tumors, with syringo-myelia, or with myelitis; it is often difficult to determine whether the case is one of primary hæmorrhage with myelitis, or myelitis with a secondary hæmorrhage.

The *anatomical condition* is very varied. The cord may be enlarged at the site of the hæmorrhage, and occasionally the white substance may be lacerated and blood may escape beneath the meninges. The extravasation is chiefly in the gray matter, and may be limited or focal, or very diffuse, extending a considerable distance in the cord. In a case which occurred at the Montreal General Hospital under Wilkins the hæmorrhage occupied a position opposite the region of the fifth and sixth cervical nerves and on transverse section the cord was occupied by a dark-red clot measuring twelve by five millimetres, around which the white substance formed a thin, ragged wall. The clot could be traced upward as far as the second cervical, and downward as far as the fourth dorsal.

The sudden onset of the *symptoms* is the most characteristic feature in hæmatomyelia. The loss of power necessarily varies with the locality affected. If in the cervical region, both arms and legs may be involved; but if in the dorsal or lumbar, there is only paraplegia. There is usually loss of sensation, and at first loss of reflexes. Myelitis frequently develops and becomes extensive, with fever and trophic changes. The condition may rapidly prove fatal; in other instances there is gradual recovery, often with partial paralysis.

The diagnosis may be made in some instances, particularly those in which the onset is sudden after injury, but there is great difficulty in differentiating hæmorrhagic myelitis from certain cases of hæmorrhage into the spinal meninges. The question of diagnosis has been carefully considered by Hoch ‡ in a recent report of two cases from my clinic.

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* Paris, 1872.

† Brain, 1889.

‡ Johns Hopkins Hospital Reports, vol. ii, fasciculus 6.

(*f*) **Caisson Disease; Diver's Paralysis.**—This remarkable affection, found in divers and in workers in caissons, is characterized by a paraplegia, more rarely a general palsy, which supervenes on returning from the compressed atmosphere to the surface.

The disease has been carefully studied by the French writers, by Leyden and Schultze in Germany, and in this country particularly by A. H. Smith. The pressure must be more than that of three atmospheres. The symptoms are especially apt to come on if the change from the high to the ordinary atmospheric pressure is quickly made. They may supervene immediately on leaving the caisson, or they may be delayed for several hours. In the mildest form there are simply pains about the knees and in the legs, often of great severity, and occurring in paroxysms. Abdominal pain and vomiting are not uncommon. The legs may be tender to the touch, and the patient may walk with a stiff gait. Dizziness and headache may accompany these neuralgic symptoms, or may occur alone. More commonly in the severe form there is paralysis both of motion and sensation, usually a paraplegia, but it may be general, involving the trunk and arms. Monoplegia and hemiplegia are rare. In the most extreme instances the attacks resemble apoplexy, and the patient rapidly becomes comatose and death occurs in a few hours. In the cases of paraplegia the outlook is usually good, and the paralysis may pass off in a day, or may continue for several weeks or even for months. Identical features are met with in the deep-sea divers.

The explanation of this condition is by no means satisfactory. Two careful autopsies have been made. In Leyden's case death occurred on the fifteenth day, and in the dorsal portion of the cord there were numerous foci of hæmorrhages and signs of an acute myelitis. In Schultze's case death occurred in two and a half months, and a disseminated myelitis was found in the dorsal region. In both cases there were fissures, and appearances as if tissue had been lacerated. It has been suggested that the symptoms are due to the liberation in the spinal cord of bubbles of nitrogen which have been absorbed by the blood under the high pressure, and the condition found at the autopsies just referred to is held to favor this view.

A large majority of the cases recover. The severe neuralgic pains often require morphia. Inhalations of oxygen and the use of compressed air have been advised. When paraplegia develops the treatment is similar to that of other forms. In all caisson work care should be exercised that the time in passing through the lock from the high to the ordinary pressure be sufficiently prolonged. According to A. H. Smith, at least five minutes should be allowed for each additional atmosphere of pressure.

III. ACUTE AFFECTIONS OF THE SPINAL CORD.

(1) Acute Diffuse Myelitis.

Etiology.—Acute myelitis results from many causes, and may affect the cord in a limited or extended portion—the gray matter chiefly, or the gray and white matter together. It is met with: (*a*) As an independent affection following exposure to cold, or exertion, and leading to rapid loss of power with the symptoms of an acute ascending paralysis. (*b*) As a sequel of the infectious diseases, such as small-pox, typhus, and measles. (*c*) As a result of traumatism, either fracture of the spine or very severe muscular effort. Concussion without fracture may produce it, but this is rare. Acute myelitis, for instance, scarcely ever follows railway accidents. (*d*) In disease of the bones of the spine, either caries or cancer. This is a more common cause of localized acute transverse myelitis than of the diffuse affection. (*e*) In disease of the cord itself, such as tumors and syphilis; in the latter, either in association with gummata, in which case it is usually a late manifestation, or it may follow within a year or eighteen months of the primary affection.*

Morbid Anatomy.—In localized acute myelitis affecting white and gray matter, as met with after accident or an acute compression, the cord is swollen, the pia injected, the consistence greatly reduced, and on incising the membrane an almost diffluent fluid may escape. In less intense grades, on section at the affected area, all trace of distinction between the gray and white matter is lost, or extremely indistinct. The tissue may be injected, or, as is often the case, hæmorrhagic. It is particularly in these forms, due to extension of disease from without or to acute compression, that we find definite involvement of the white matter. In other instances the gray matter is chiefly affected. There may be localized areas throughout the cord in which the gray matter is reduced in consistence and hæmorrhagic, the so-called red softening. There may be definite cavity formations in these foci. In some cases of disseminated or focal myelitis the meninges also are involved and there is a myelo-meningitis. And, lastly, there are instances in which, throughout a long section of the cord, sometimes through the lumbar and the greater part of the dorsal, or in the dorsal and cervical regions, there is a diffuse myelitis of the gray substance.

Histologically the nerve fibres are much swollen and irregularly distorted, the axis cylinders are beaded, the myelin droplets are abundant, and the laminated bodies known as corpora amylacea may be seen. The granular fatty cells are also numerous and there may be leucocytes and red blood-corpuscles. Changes in the blood-vessels are striking; the smaller veins are distended and may show varicosities. The perivascular

* Breteau, Des Maladies Syphilitiques Précoces, Paris Thesis, 1889.

lymph spaces contain numerous leucocytes, and the smaller arteries themselves are frequently the seat of hyaline thrombi. The ganglion cells are swollen and irregular in outline, the protoplasm is extremely granular and vacuolated, and the nuclei, though usually invisible, may show signs of division, and the processes of the cells are not seen.

In cases which persist for some time we have an opportunity of seeing the later stages of acute myelitis. The acute, inflammatory, hyperæmic or red softening is succeeded by stages in which the affected area becomes more yellow from gradual alteration of the blood-pigment, and finally white in color from the advancing fatty degeneration. In cases of compression myelitis, a sclerosis may gradually be produced with the anatomical picture of a chronic diffuse myelitis.

Symptoms.—(*a*) *Acute Central Myelitis.*—It is this form which comes on spontaneously after cold, or in connection with syphilis or one of the infectious diseases, or is seen in a typical manner in the extension from injuries or from tumor. The onset, though scarcely so abrupt as in hæmorrhage, may be sudden; a person may be attacked on the street and have difficulty in getting home. In some instances, the onset is preceded by pains in the legs or back, or a girdle sensation is present. It may be marked by chills, occasionally by convulsions; fever is usually present from the beginning—at first slight, but subsequently it may become high.

The *motor* functions are rapidly lost, sometimes as quickly as in Landry's ascending paralysis. The paraplegia may be complete, and, if the myelitis extends to the cervical region, there may be impairment of motion, and ultimately complete loss of power of the upper extremities as well. The sensation is lost, but there may at first be hyperæsthesia. The reflexes in the initial stage are increased, but in acute central myelitis, unless limited in extent to the dorsal and cervical regions, the reflexes are usually abolished. The rectum and bladder are paralyzed. Trophic disturbances are marked; the muscles waste rapidly; the skin is often congested, and there may be localized sweating. The temperature of the affected limbs may be lowered. Acute bed-sores may develop over the sacrum or on the heels, and sometimes a multiple arthritis is present. In these acute cases the general symptoms become greatly aggravated, the pulse is rapid, the tongue becomes dry; there is delirium, the fever increases, and may reach 107° or 108°.

The course of the disease is variable. In very acute cases death follows in from five to ten days. The cases following the infectious diseases particularly the fevers and sometimes syphilis, may run a milder course.

The diagnosis of this variety of acute myelitis is rarely difficult. In common with the acute ascending paralysis of Landry, and with certain cases of multiple neuritis, it presents a rapid and progressive motor paralysis. From the former it is distinguished by the more marked involvement of sensation, the trophic disturbances, the paralysis of bladder and rectum,

the rapid wasting, the electrical changes, and the fever. From acute cases of multiple neuritis it may be more difficult to distinguish, as the sensory features in these cases may be marked, though there is rarely, if ever, in multiple neuritis complete anæsthesia; the wasting, moreover, is more rapid in myelitis. The bladder and rectum are rarely involved—though in exceptional cases they may be—and, most important of all, the trophic changes, the development of bullæ, bed-sores, etc., are not seen in multiple neuritis.

(*b*) *Acute Transverse Myelitis.*—The symptoms naturally differ with the situation of the lesion.

(1) Acute transverse myelitis in the *dorsal region*, the most common situation, produces a very characteristic picture. The symptoms of onset are variable. There may be initial pains or numbness and tingling in the legs. The paralysis may set in quickly and become complete within a few days; but more commonly it is preceded for a day or two by sensations of pain, heaviness, and dragging in the legs. The paralysis of the lower limbs is usually complete, and if at the level, say, of the sixth dorsal vertebra, the abdominal muscles are involved. Sensation may be partially or completely lost. At the onset there may be numbness, tingling, or even hyperæsthesia in the legs. At the level of the lesion there is often a zone of hyperæsthesia, which is discovered by passing a test-tube containing hot water along the spine, when the sensation of warmth changes to one of actual pain. A girdle sensation may occur early, and when the lesion is in this situation it is usually felt between the ensiform and umbilical regions. The reflex functions are variable. There may at first be abolition of the reflexes; subsequently, the reflexes, passing through the segments lower than the one affected, may be exaggerated and the limbs may pass into a condition of spastic rigidity. It does not always happen, however, that the reflexes are increased in a total transverse lesion of the cord. They may be entirely lost, as pointed out some years ago by Bastian, and insisted upon by him in a recent memoir.* F. T. Miles has also called attention to this fact and reported five cases in which the reflexes were lost in total transverse lesion of the cord. That this is not due to the preliminary shock is shown by the fact that the abolition of the reflexes may continue for four or more months. The trophic changes are not marked. The muscles become extremely flabby, but not wasted in an extreme degree; subsequently rigidity develops. If the gray matter of the lumbar cord is involved, the flaccidity persists and the wasting may be considerable. The reaction of degeneration is not present. The temperature of the paralyzed limbs is variable. It may at first rise, then fall and become subnormal. Lesions of the skin are not uncommon, and bed-sores are apt to form. There is at first retention of urine and subsequent incontinence. If the lumbar centres are involved, there are from the outset vesical symptoms. The urine is alka-

* Medico-Chirurgical Transactions, vol. lxxiii.

line in reaction and may rapidly become ammoniacal. The bowels are constipated and there is usually incontinence of the fæces. Some writers attribute the cystitis associated with transverse myelitis to disturbed trophic influence.

The course of complete transverse myelitis depends a good deal upon its cause. Death may result from extension. Segments of the cord may be completely and permanently destroyed, in which case there is persistent paraplegia. The pyramidal fibres below the lesion undergo the secondary degeneration, and there is an ascending degeneration of the posterior median columns. If the lower segments of the cord are involved the legs may remain flaccid. In some instances a transverse myelitis of the dorsal region involves the anterior horns above and below the lesion, producing flaccidity of the muscles, with wasting, fibrillar contractions, and the reaction of degeneration. More commonly, however, in the cases which last many months there is more or less rigidity of the muscles with spasm or persistent contraction of the flexors of the knee.

(2) *Transverse Myelitis of the Cervical Region.*—If at the level of the sixth or seventh cervical nerves, there is paralysis of the upper extremities, more or less complete, sometimes sparing the muscles of the shoulder. Gradually there is loss of sensation. The paralysis is usually complete below the point of lesion, but there are rare instances in which the arms only are affected, the so-called cervical paraplegia. In addition to the symptoms already mentioned there are several which are more characteristic of transverse myelitis in the cervical region, such as the occurrence of vomiting, hiccough, and slow pulse, which may sink to twenty or thirty, pupillary changes—myosis—sometimes attacks of dysphagia, dyspnœa, or syncope.

II. Myelitis of the Anterior Horns

(*Polio-myelitis Anterior; Atrophic Spinal Paralysis*).

Definition.—An affection occurring most commonly within the first three years of life, characterized by fever, loss of power in certain muscles, and rapid atrophy.

Etiology.—The cause of the disease is unknown. It has been attributed to cold, to the irritation from dentition, or to overexertion. Since the days of Mephibosheth, parents have been inclined to attribute this form of paralysis to the carelessness of nurses in letting the children fall, but very rarely is the disease induced by traumatism, and in perhaps a majority of the cases the child is attacked while in full health. As Sinkler has pointed out, the cases are more common in the warm months. Boys are more liable to be affected than girls. Several instances of the occurrence of numerous cases together in epidemic form have been described. Medin reports from Stockholm an epidemic in which from the 9th of August to the 23d of September 29 cases came under observation. In two instances two children in the same family were attacked within a few days.

Although most frequent in children, it develops occasionally in young adults, or even in middle-aged persons.

Morbid Anatomy.—The disease is oftenest seen in either the cervical or lumbar enlargements. In very early cases, such as those described by David Drummond and Charlewood Turner, the lesion has been that of an acute hæmorrhagic myelitis with degeneration and rapid destruction of the large ganglion cells. The condition may be strictly confined to the anterior cornua; in some instances there is slight meningeal involvement. In cases in which the examination is not made for some months or years the changes are very characteristic. The anterior cornu in the affected region is greatly atrophied and the large motor cells are either entirely absent or only a few remain. The affected half of the cord may be considerably smaller than the other. The antero-lateral column may show slight sclerotic changes, chiefly in the pyramidal tract. The corresponding anterior nerve roots are atrophied, and the muscles are wasted and gradually undergo a fatty and sclerotic change.

Symptoms.—In a majority of the cases, after slight indisposition and feverishness, the child is noticed to have lost the use of one limb. Convulsions at the outset are rare, not constant as in the acute cerebral palsies of children. Fever is usually present, the temperature rising to 101°, sometimes to 103°. Pain is rarely complained of; there may occasionally be slight aching in the joints. The paralysis is abrupt in its onset and, as a rule, is not progressive, but reaches its maximum in a very short time, even within twenty-four hours. It is rarely generalized. The suddenness of onset is remarkable and suggests a primary affection of the blood-vessels, a view which the hæmorrhagic character of the early lesion supports. The distribution of the paralysis is very variable. One or both arms may be affected, one arm and one leg, or both legs; or it may be crossed paralysis, the right leg with the left arm. In the upper extremities the paralysis is rarely complete and groups of muscles may be affected. As Remak has pointed out, there is an upper-arm and a lower-arm type of palsy. The deltoid, the biceps, brachialis anticus, and supinator longus may be affected in the former, and in the latter the extensors or flexors of the fingers and wrists. This distribution is due to the fact that the groups of nerve-cells are attacked which preside over certain muscles acting functionally together.

In the legs the tibialis anticus and extensor groups of muscles are more affected than the hamstrings and glutei. The muscles of the face are never, the sphincters rarely, involved. While the rule is for the paralysis to be abrupt and sudden, there are cases in which it comes on slowly and takes from three to five days for its development. At first the affected limb looks natural, and as children between two and three are usually fat, very little change may be noticed for some time; but the atrophy proceeds rapidly, and the limb becomes flaccid and feels soft and flabby. Usually as early as the end of the first week the reaction of degeneration

is present. The nerves are found to have lost their irritability. The muscles do not react to the induced current, but to the constant current they respond by a sluggish contraction, usually to a weaker current than is normal, and more to the positive pole than to the negative. The paralysis remains stationary for a time, and then there is gradual improvement. Complete recovery is rare, and, when the anatomical condition is considered, is scarcely to be expected. The large motor cells of the cornua, when thoroughly disintegrated, cannot be restored. In too many cases the improvement is only slight and permanent paralysis remains in certain groups. Sensation is unaffected; the skin reflexes are absent, and the deep reflexes are usually lost.

When the paralysis persists the wasting is extreme, the growth of the bones of the affected limb is arrested, or at any rate retarded, and the joints may be very relaxed; as, for instance, when the deltoid is affected the head of the humerus is no longer kept in contact with the glenoid cavity. In the later stages very serious deformities are produced by the contracture of the muscles.

Diagnosis.—The condition is only too evident in the majority of cases. There is a flaccid, flabby paralysis of one or more limbs which has set in abruptly. The rapid wasting, the lax state of the muscles, the electrical reactions, and the absence of reflexes distinguish it from the cerebral palsies. The pseudo-paresis of rickets is a condition to be carefully distinguished. In this the loss of power is in the legs, rapid atrophy is not present, certain movements are possible but painful. The general hyperæsthesia of the skin, the characteristic changes in the bones, and the diffuse sweats are present. Disease of the hip or knee may produce a pseudo-paralysis which can with care be readily distinguished.

Prognosis.—The outlook in any case for complete recovery is bad. The natural course of the disease must be borne in mind; the sudden onset, the rapid but not progressive loss of power, a stationary period, then marked improvement in certain muscle groups, and finally in many cases contractures and deformities. There is no other disease in which the physician is so often subject to unjust criticism, and the friends should be told at the outset that in the severe and extensive paralysis complete recovery should not be expected. The best to be hoped for is a gradual restoration of power in certain muscle groups. In estimating the probable grade of permanent paralysis, the electrical examination is of great value.

Treatment of Acute Myelitis.—In the rapidly developing form due either to a diffuse inflammation in the gray matter or to transverse myelitis, the important measures are: Scrupulous cleanliness, care and watchfulness in guarding against bed-sores, the avoidance of cystitis, either by systematic catheterization or, if there is incontinence, by a carefully adjusted bed urinal, or the use of antiseptic cotton-wool repeatedly changed. In an acute onset in a healthy subject the spine may be cupped. Counter-irritation is of doubtful advantage. Chapman's ice-bag is some-

times useful. No drugs have the slightest influence upon an acute myelitis, and even in subjects with well-marked syphilis neither mercury nor iodide of potassium is curative. Tonic remedies, such as quinine, arsenic, and strychnia, may be used in the later stages. When the muscles have wasted, massage is beneficial in maintaining their nutrition. Electricity should not be used in the early stages of myelitis. It is of no value in the transverse myelitis in the dorsal region with retention of the nutrition in the muscles of the leg.

The treatment of acute infantile paralysis has a bright and a dark side. In a case of any extent complete recovery cannot be expected; on the other hand, it is remarkable how much improvement may finally take place in a limb which is at first completely flaccid and helpless. The following treatment may be pursued: If seen in the febrile stage, a brisk laxative and a fever mixture may be given. The child should be in bed and the affected limb or limbs wrapped in cotton. As in the great majority of cases the damage is already done when the physician is called and the disease makes no further progress, the application of blisters and other forms of counter-irritation to the back is irrational and only cruel to the child.

The general nutrition should be carefully maintained by feeding the child well, and taking it out of doors every day. As soon as the child can bear friction the affected part should be carefully rubbed; at first once a day, subsequently morning and evening. Any intelligent mother can be taught systematically to rub, knead, and pinch the muscles, using either the bare hand or, better still, sweet oil or cod-liver oil. This is worth all the other measures advised in the disease, and should be systematically practised for months, or even, if necessary, a year or more. Electricity has a much more limited use, and cannot be compared with massage in maintaining the nutrition of the muscles. The faradic current should be applied to those muscles which respond. The essence of the treatment is in maintaining the nutrition of the muscles, so that in the gradual improvement which takes place in parts, at least, of the affected segments of the cord the motor impulses may have to deal with well-nourished, not atrophied muscle fibres.

Of medicines, in the early stage ergot and belladonna have been warmly recommended, but it is unlikely that they have the slightest influence. Later in the disease strychnia may be used with advantage in one or two minim doses of the liquor strychninæ, which, if it has no other effect, is a useful tonic.

The most distressing cases are those which come under the notice of the physician six, eight, or twelve months after the onset of the paralysis, when one leg or one arm or both legs are flaccid and have little or no motion. Can nothing be done? A careful electrical test should be made to ascertain which muscles respond. This may not be apparent at first, and several applications may be necessary before any contractility is

noticed. With a few lessons an intelligent mother can be taught to use the electricity as well as to apply the massage. If in a case in which the paralysis has lasted for six or eight months no observable improvement takes place in the next six months with thorough and systematic treatment, little or no hope can be entertained of further change.

In the later stage care should be taken to prevent the deformities resulting from the contractions. Great benefit results from a carefully applied apparatus.

III. Acute and Subacute Polio-myelitis in Adults.

An acute polio-myelitis in adults, the exact counterpart of the disease in children, is recognized. A majority, however, of the cases described under this heading have been multiple neuritis; but the suddenness of onset, the rapid wasting, and the marked reaction of degeneration are thought by some to be distinguishing features. Multiple neuritis may, however, set in with rapidity; there may be great wasting and the reaction of degeneration is sometimes present. The time element alone may determine the true nature. Recovery in a case of extensive multiple paralysis from polio-myelitis will certainly be with loss of power in certain groups of muscles; whereas, in multiple neuritis the recovery, while slow, may be perfect.

The subacute form, the *paralysie générale spinale antérieure subaiguë* of Duchenne, is in all probability a peripheral palsy. The paralysis usually begins in the legs with atrophy of the muscles, then the arms are involved, but not the face. Sensation is, as a rule, not involved.

IV. Acute Ascending (Landry's) Paralysis.

Definition.—An advancing paralysis, beginning in the legs, rapidly extending to the trunk and arms, and finally, in many cases, involving the muscles of respiration. It presents a remarkable similarity in its symptoms to certain cases of polyneuritis, with which it is now grouped by many writers.

Etiology and Pathology.—The disease occurs most commonly in males between the twentieth and thirtieth years. It has sometimes followed the specific fevers. An elaborate study of 93 cases collected from the literature has been made by James Ross, who concludes that in etiology, symptoms, course, and termination it conforms to a peripheral neuritis. Neuwerk and Barth have reached a similar conclusion. In their case an interstitial neuritis was found in the nerve roots, but the peripheral nerves were normal. On the other hand, cases have been reported of rapidly ascending paralysis in which the periphral nerves and nerve roots were unaffected. In a case of eleven days' duration recently studied by Hun, the lesions were certainly too slight to account for the advancing and wide-spread paralysis, and, with our present knowledge, Hun is cor-

rect in stating that "acute ascending paralysis—defined so as to exclude all cases in which the sensory symptoms are prominent, or in which well-marked bulbar symptoms are not present—must therefore be regarded as a clinical entity for which no corresponding lesion has as yet been discovered." It is not improbable that some toxic agent is responsible for the symptoms.

Symptoms.—Weakness of the legs, gradually progressing, often with tolerable rapidity, is the first symptom. In some cases within a few hours the paralysis of the legs becomes complete. The muscles of the trunk are next affected, and within a few days, or even less in more acute cases, the arms are also involved. The neck muscles are next attacked, and finally the muscles of respiration, deglutition, and articulation. The reflexes are lost, but the muscles neither waste nor show electrical changes. The sensory symptoms are variable; in some cases tingling, numbness, and hyperæsthesia have been present. In the more characteristic cases sensation is intact and the sphincters are uninvolved. Enlargement of the spleen has been noticed in several cases. The course of the disease is variable. It may prove fatal in less than two days. Other cases persist for a week or for two weeks. In some instances recovery has occurred, but in a large proportion of the cases the disease is fatal.

The *diagnosis* is difficult, particularly from certain forms of multiple neuritis, and if we include in Landry's paralysis the cases in which sensation is involved, distinction between the two affections is impossible. We apparently have to recognize the existence of a rapidly advancing motor paralysis without involvement of the sphincters, without wasting or electrical changes in the muscles, without trophic lesions, and without fever—features sufficient to distinguish it from either the acute central myelitis or the polio-myelitis anterior. It is doubtful, however, whether these characters always suffice to enable us to differentiate the cases of multiple neuritis.

IV. CHRONIC AFFECTIONS OF THE SPINAL CORD.

I. Spastic Paraplegia.

Definition.—Loss of power with spasm of the muscles of the lower extremities.

While clinically spastic paraplegia, or, as it is sometimes called, *tabes dorsalis spasmodique*, is a well-defined, readily recognizable affection, etiologically and anatomically it presents marked differences, and various groups must be separated, all of which present, however, the combination of spasm with loss of power. As the pyramidal tracts are involved the term lateral sclerosis is sometimes used as the equivalent of spastic paraplegia. I shall consider the following forms:

(1) **Secondary Spastic Paralysis.**—After any tranverse lesion of the cord, whether the result of slow compression (as in caries), chronic myelitis, the pressure of tumor, chronic meningo-myelitis, or multiple sclerosis, degeneration takes place in the pyramidal tracts below the point of disease. The legs soon become stiff and rigid, and the reflexes increase. It happens occasionally, as Bastian has shown, that in compression paraplegia the limbs may be flaccid without increase in the reflexes—*paraplegie flasque* of the French. The condition of the patient in these secondary forms varies very much. In chronic myelitis or in multiple sclerosis he may be able to walk about, but with a characteristic spastic gait. In the compression myelitis, in fracture, or in caries, there may be complete loss of power with rigidity.

(2) **Primary Spastic Paraplegia.**—This is believed to depend upon a primary sclerosis of the lateral or pyramidal tracts. Clinically it is common to meet with cases in adults, particularly in syphilitic subjects, who have pains in the back, perhaps a girdle sensation, and a gradually developing, progressive spastic paraplegia. It may be impossible from the history or the physical examination to determine whether the condition is secondary to a transverse myelitis or a meningo-myelitis, or whether the lesion is a primary degeneration of the pyramidal tracts. The question is still debated whether a primary lesion of the lateral tracts ever takes place, or whether, in such instances, there is not always some lesion of the motor cells in the anterior horns. Cases may persist for years without any atrophy. In other instances there are signs of involvement of the posterior columns as well, forming the condition of ataxic paraplegia, which will be considered separately. So far as I know, the only case which is claimed to demonstrate the existence of a primary lateral sclerosis is that of Dreschfeld's, which occurred in 1881.

The *symptoms* of spastic paraplegia are very distinctive. The patient complains of feeling tired, of stiffness in the legs, and perhaps of pains of a dull aching character in the back or in the calves. There may be no definite loss of power, even when the spastic condition is well established. In other instances there is definite weakness. The stiffness is felt most in the morning. In a well-developed case the gait is most characteristic. The legs are moved stiffly and with hesitation, the toes drag and catch against the ground, and, in extreme cases, when the ball of the foot rests upon the ground a distinct clonus develops. The legs are kept close together, the knees touch, and in certain cases the adductor spasm may cause cross-legged progression. On examination, the legs may at first appear tolerably supple, perhaps flexed and extended readily. In other cases the rigidity is marked, particularly when the limbs are extended. The spasm of the adductors of the thigh may be so extreme that the legs are separated with the greatest difficulty. In cases of this extreme rigidity the patient usually loses the power of walking. The nutrition is well maintained, the muscles may be hypertrophied. The reflexes are greatly

increased. The slightest touch upon the patellar tendon produces an active knee-jerk. The rectus clonus and the ankle clonus are easily obtained. In some instances the slightest touch may throw the legs into violent clonic spasm, the condition to which Brown-Séquard gave the name of spinal epilepsy. The superficial reflexes are also increased. The arms may be unaffected for years, but as a late manifestation rigidity may develop.

The *diagnosis* is readily made, but it is often very difficult to determine accurately the nature of the underlying pathological condition. A history of syphilis is present in many of the cases. The course of the disease is progressively downward. Years may elapse before the patient is bedridden. Involvement of the sphincters, as a rule, is late; occasionally, however, it is early. The sensory symptoms rarely progress and the patients may retain the general nutrition and enjoy excellent health. Ocular symptoms are rare.

(3) **The Spastic Paraplegia of Infants** (*Paraplegia Cerebralis Spastica* —Heine).—This is usually a birth palsy, often the result of difficult labor. In twenty-three of the twenty-four of Little's cases, there was either difficult labor or premature delivery. Several children may be affected in a family. Gee reports two cases in one family, Schultze three, and with Latimer I saw a brother and a sister with the disease. In this connection it is interesting to note that Bernhardt has recently described a family form of spastic paraplegia, in which four brothers were affected, the disease developing in each about the thirtieth year. The stiffness of the legs may not be noticed for some months after birth, but usually on dressing the child the mother notices the rigidity. When attempts are made to walk the stiffness and awkwardness then become apparent. On standing, the attitude is very characteristic. There is talipes equinus, varying from the slightest raising of the heel to a condition in which the child stands on tiptoe. In older children, as they walk, the toe-cap of the shoe is usually much worn. The strong adductor action may produce typical cross-legged progression, in which each foot is dragged over and planted in front, or even on the other side of its fellow. In attempting to flex the legs there is a marked resistance, which gradually yields—the lead-pipe contraction, as Weir Mitchell calls it. The reflexes are increased, though in some children it is not an easy matter to obtain them. The ankle clonus, as a rule, is not obtainable. Sensation is unimpaired, and the bladder and rectum are not involved.

The symptoms of this affection in children are almost identical with the spastic paraplegia of adults. The arms may be involved—spastic diplegia. The disease is probably of cortical origin. There are frequently symptoms indicating cerebral defects, such as idiocy, imbecility, and nystagmus. Some of the cases depend, no doubt, upon bilateral meningeal hæmorrhage occurring during delivery. Others are probably due to arrest of development of the pyramidal tracts. This condition in children must

not be confounded with tetany or with the pseudo-paralytic rigidity so often associated with rickets.

(4) **Ataxic Paraplegia.**—This name is applied by Gowers to a disease characterized clinically by a combination of ataxia and spastic paraplegia, and anatomically by involvement of the posterior and lateral columns.

The disease is most common in middle-aged males. Exposure to cold and traumatism have been occasional antecedents. In striking contrast to ordinary tabes a history of syphilis is rarely to be obtained.

The anatomical features are a sclerosis of the posterior columns, which is not more marked in the lumbar region and not specially localized in the root zone of the postero-external columns. The involvement of the lateral columns is diffuse, not always limited to the pyramidal tracts, and there may be an annular sclerosis.

The *symptoms* are well defined. The patient complains of a tired feeling in the legs, not often of actual pain. The sensory symptoms of true tabes are absent. An unsteadiness in the gait gradually develops with progressive weakness. The reflexes are increased from the outset, and there may be well-developed ankle clonus. Rigidity of the legs slowly comes on, but is rarely so marked as in the uncomplicated cases of lateral sclerosis. From the start, incoördination is a well-characterized feature, and the difficulty of walking in the dark or swaying when the eyes are closed may, as in true tabes, be the first symptom to attract attention. In walking the patient uses a stick, keeps the eyes fixed on the ground, the legs far apart, but the stamping gait, with elevation and sudden descent of the feet, is not often seen. The incoördination may extend to the arms. Sensory symptoms are rare, but Gowers calls attention to a dull, aching pain in the sacral region. The sphincters usually become involved. Eye symptoms are rare. Late in the disease mental symptoms may develop, similar to those of general paresis.

In well-marked cases the *diagnosis* is easy. The combination of marked incoördination with retention of the reflexes and more or less spasm are characteristic features. The absence of ocular and sensory symptoms is an important point.

(5) **Hysterical Spastic Paraplegia.**—There is no spinal-cord disease which may be so accurately mimicked by hysterical patients as spastic paraplegia. There is wasting in the hysterical paraplegia, the sensory symptoms are not marked, the loss of power is not complete, and there is not that extensor spasm so characteristic of organic disease. The hysterical contracture will be considered later.

The reflexes are, as a rule, increased. The knee-jerk is present, and there may be well-developed ankle clonus. Gowers calls attention to the fact that it is usually a spurious clonus, "due to a half-voluntary contraction in the calf muscles." A true clonus does occur, however, and there may be the greatest difficulty in determining whether or not the case is one of hysterical paraplegia.

(6) **Primary Combined Sclerosis (Putnam).**—In addition to the ataxic paraplegia just mentioned, here may be considered certain cases which are characterized anatomically by a relatively chronic sclerosis of the posterior columns, of the lateral columns, chiefly the pyramidal tract, and also of the cerebellar tract. With these are usually associated more acute changes in adjoining areas, either diffuse or systemic, some grade of degeneration in the gray matter, and involvement of the nerve roots. This form has been studied by J. J. Putnam and Dana. The cases are usually in women —seven out of nineteen collected by Dana; the ages, from forty-five to sixty-four. The disease runs a rather rapid course. Neuropathic inheritance is present in some instances. Putnam thinks that possibly both lead and arsenic play a part in the etiology.

The *symptoms* are both sensory and motor. The onset is usually with numbness in the extremities, progressive loss of strength, and emaciation. Paraplegia gradually develops, before which there have been, as a rule, spastic symptoms with exaggerated knee-jerk. The arms are affected less than the legs. Mental symptoms similar to dementia paralytica may develop toward the close.

The *diagnosis* of this mixed sclerosis rests upon the combination of sensory and motor symptoms with the presence of exaggerated reflexes. As stated, the sensory features consist chiefly of paræsthesia, and there may be difficulty in distinguishing the condition from multiple neuritis. The frequency of the disease in more or less enfeebled or anæmic women past middle life is also an important feature.

Treatment of Spastic Paraplegia.—In the majority of cases spastic paraplegia is incurable. The cases which result from transitory compression, as in caries, may get well; but in the other forms the disease is uniformly progressive, and remedies have little or no control. When syphilis is suspected a thorough course of mercury and iodide of potassium should be given. Scrupulous attention should be paid to the bladder symptoms, and the same measures may be used as will be advised in locomotor ataxia. In the infantile form of paraplegia much may be done by the orthopædic surgeon to overcome rigidity and contracture. In several instances I have known persistent friction with forcible flexion and extension and the application of proper apparatus enable a patient to get about comfortably.

II. Locomotor Ataxia

(*Tabes Dorsalis; Posterior Spinal Sclerosis*).

Definition.—An affection of the nervous system characterized clinically by incoördination, with sensory and trophic disturbances and involvement of the special senses, particularly the eyes. Anatomically there are found sclerosis of the posterior columns of the cord, foci of degeneration in the basal ganglia, and sometimes chronic degenerative changes in the cortex cerebri.

Etiology.—It is a wide-spread disease, more frequent in cities than in the country. The relative proportion may be judged from the fact that of 1,816 cases in my neurological dispensary in two years there were 25 cases of locomotor ataxia. Males are attacked more frequently than females, the proportion being at least ten to one. Mitchell has called attention to the fact that it is a rare disease in the negro. Of 25 cases at my clinic, 3 were in negroes. It is a disease of adult life, a majority of the cases occurring between the thirtieth and fortieth years. Occasionally cases are seen in young men. The form of ataxia which occurs in children is a different disease. Of special causes syphilis is the most important. According to the figures of Erb, Fournier, and Gowers, in from fifty to seventy-five per cent of all cases there is a history of this disease. Erb's recent figures are most striking; of 300 cases of tabes in private practice 89 per cent had had syphilis.

Excessive fatigue, overexertion, exposure to cold and wet, and sexual excesses are all assigned as causes. There are instances in which the disease has closely followed severe exposure. James Stewart has noted that the Ottawa lumbermen, who live a very hard life in the camps during the winter months, are frequently the subjects of locomotor ataxia. Trauma has been noted in a few cases. Alcoholic excess does not seem to predispose to the disease. Among patients in the better classes of life I do not remember one in which there had been a previous history of prolonged drunkenness.

Morbid Anatomy.—When a patient has died in the advanced stage of the disease the following are the most important changes:

(*a*) The peripheral nerves may show signs of degeneration. Neuritis may indeed be present even when there have been no special symptoms indicating it. In other instances there is not only neuritis, but muscular atrophy.

(*b*) The posterior roots of the spinal cord are small, gray, and atrophic.

(*c*) The meninges of the posterior and lateral columns are thickened, more firmly adherent than normally, and the blood-vessels usually show signs of arterio-sclerosis.

(*d*) The changes in the spinal cord are as follows: (1) In advanced cases the posterior columns are uniformly sclerotic and the dorsal and lumbar regions are most extensively involved. In long-standing cases there is generally an increase of connective tissue throughout the cord and there may be degeneration (2) of the ascending antero-lateral tract; (3) of the direct cerebellar tract; (4) of the pyramidal tract.

(*e*) In early cases the course of the anatomical changes may be traced. The steps in the process are as follows: The posterior root-zone of Charcot is first involved, often with the fibres of the posterior root, so that it has been thought to begin perhaps as a neuritis of these roots within the vertebral canal. The narrow strip which lies between the pyramidal tract

and the posterior cornu, known as Lissauer's tract, is early involved, together with the nerve-cells of the adjacent Clarke's vesicular column. In what is known as the pre-ataxic stage these may be the only alterations. Subsequently the sclerosis extends widely in the postero-external, and subsequently in the postero-median columns.

(*f*) The cerebral changes—of less consequence than the spinal—may consist of (1) sclerosis in the restiform bodies, in the inferior peduncles of the cerebellum, and of certain of the cranial nerves, particularly the third, the optic, and the auditory; (2) cortical changes, consisting in some cases of a diffuse meningo-encephalitis.

Symptoms.—These are best considered under the three stages of pre-ataxic, ataxic, and paralytic.

Pre-ataxic Stage.—The following are the most characteristic features of this period:

Pains, usually of a sharp stabbing character; hence the term, lightning pains. They last for only a second or two and are most common in the legs. They may be associated with a hot, burning feeling. Occasionally herpes may develop at the site of the pain. They may occur at irregular intervals, and are more prone to follow excesses or to come on when the health is impaired.

Ocular Symptoms.—(*a*) Ptosis, which may be single or double and is by no means uncommon either alone or (*b*) in association with external strabismus. The first complaint may be of double vision. Occasionally there may be paralysis of all the external muscles of the eye, producing ophthalmoplegia externa. (*c*) Argyll-Robertson pupil, in which, as already mentioned, there is loss of the iris reflex to light, but contraction during accommodation. The pupils are usually small—spinal myosis. (*d*) Optic atrophy. This is often an early, or even the first symptom. The loss of vision progresses, and in a large majority of cases leads to total blindness.

Loss of the Knee-jerk.—This is one of the earliest symptoms, and may occur years before there is ataxia. Taken alone it is of no moment, as there are individuals in whom the knee-jerk is absent; but in connection with the lightning pains and the ocular symptoms, it is of special importance. These are the most common symptoms of the pre-ataxic stage, and may persist for years without the development of incoördination. The patient may look well and feel well, and be troubled only by occasional attacks of lightning pains; or there is persistent ptosis, external strabismus develops, or, what is more serious, a progressive atrophy of the optic nerve. There is often a gradual loss of sexual power.

The disease may never progress beyond this stage, and when optic atrophy develops early and leads to blindness, the ataxia rarely, if ever, supervenes. There is a sort of antagonism between the ocular symptoms and the progress of the ataxia. Charcot lays considerable stress upon this, and Déjérine assured me that of the enormous tabetic material at the

Bicêtre in not a single instance in which optic atrophy had come on early and progressed to blindness was the patient ataxic, although there were cases which had had the lightning pains and lesions of the optic nerves for twenty-five years.

Ataxic Stage. — *Motor Symptoms.*—The ataxia develops gradually. One of the first indications to the patient is inability to get about readily in the dark or to maintain his equilibrium when washing his face with the eyes shut. When the patient stands with the feet together and the eyes closed, he sways and has difficulty in maintaining his position. This is known as Romberg symptom. On turning quickly he is apt to fall. Gradually the characteristic ataxic gait develops. The patient, as a rule, walks with a stick, the eyes are directed to the ground, the body is thrown forward, and the legs are wide apart. In walking, the leg is thrown out violently, the foot is raised too high and is brought down in a stamping manner with the heel first, or the whole sole comes in contact with the ground. Ultimately the patient may be unable to walk without the assistance of two canes. This gait is very characteristic, and unlike that seen in any other disease. The incoördination is not only in walking, but in the performance of other movements. If the patient is asked, when in the recumbent posture, to touch the knee with one foot, the irregularity in the movement is very evident. Incoördination of the arms is less common, but usually develops in some grade. It may in rare instances exist before the incoördination of the legs. In the large number of ataxics which frequented the Infirmary for Nervous Diseases at Philadelphia, there was only one, so far as I remember—at Weir Mitchell's clinic—in which the arms were first affected. It may be tested by asking the patient to close his eyes and to touch the tip of the nose or the tip of the ear with the finger, or with the arms thrust out to bring the tips of the fingers together. The incoördination may early be noticed by a difficulty which the patient experiences in buttoning his collar or in performing one of the ordinary routine acts of dressing.

One of the most striking features of the disease is that with marked incoördination there is no loss of muscular power. The grip of the hands may be strong and firm, the power of the legs, tested by trying to flex them, may be unimpaired, and their nutrition, except toward the close, may be unaffected.

Sensory Symptoms.—The lightning pains may persist. They vary greatly in different cases. Some patients are rendered miserable by the frequent occurrence of the attacks; others escape altogether. In addition, common symptoms are tingling, pins and needles, particularly in the feet, and areas of hyperæsthesia or of anæsthesia. The patient may complain of a change in the sensation in the soles of the feet, as if cotton was interposed between the floor and the skin. Sensory disturbances occur less frequently in the hands. Retardation of tactile sensation is common, and a pin-prick on the foot, instead of being instantaneously felt, is not per-

ceived for a second or two or may be delayed for as much as ten seconds. The pain felt may persist. A curious phenomenon is the loss of the power of localizing the pain. For instance, if the patient is pricked on one limb he may say that he feels it on the other (allocheiria), or a pin-prick on the foot may be felt in both feet. The muscular sense becomes much impaired and the patient no longer recognizes the position in which his limbs are placed. This may be present in the pre-ataxic stage.

Reflexes.—As mentioned, the loss of the knee-jerk is one of the earliest symptoms of the disease. Occasionally a case is found in which it is retained. The skin reflexes may at first be increased, but later are usually involved with the deep reflexes.

Special Senses.—The eye symptoms noted above may be present, but, as mentioned, ataxia is rare with atrophy of the optic nerve.

Deafness may develop, due to lesion of the auditory nerve. There may also be attacks of vertigo. Olfactory symptoms are rare.

Visceral Symptoms.—Among the most remarkable sensory disturbances are the tabetic crises, severe paroxysms of pain referred to various viscera; thus laryngeal, gastric, nephralgic, rectal, urethral, and clitoral crises have been described. The most common are the gastric and laryngeal. In the former there are intense pains in the stomach, vomiting, and a secretion of hyperacid gastric juice. The attack may last for several days or even longer. There may be severe pain without any vomiting. The attacks are of variable intensity and usually require morphia. Paroxysms of rectal pain and tenesmus are described. They have not been common in my experience. Laryngeal crises also are rare. There may be true spasm with dyspnœa and noisy inspiration. In one instance at least the patient has died in the attack.

The sphincters are frequently involved. Early in the disease there may be a retardation or hesitancy in making water. Later there is retention, and cystitis may occur. Unless great care is taken the inflammation may extend to the kidneys. Constipation is extremely common. Late in the disease the sphincter ani is weakened. The sexual power is usually lost in the ataxic stage.

Trophic Changes.—Skin rashes may develop in the course of the lightning pains, such as herpes, œdema, or local sweating. Alteration in the nails may occur. A perforating ulcer may develop on the foot, usually beneath the great toe. Onychia may prove very troublesome.

The arthropathies or joint lesions affect chiefly the knees. They are unquestionably associated with the disease itself, and not necessarily a result of trauma. The condition, known as Charcot's joint, is anatomically similar to that of chronic arthritis deformans. The effusion may be rapid and there may be great disintegration and destruction of the cartilages and bones, leading to dislocation and deformity. Pus was present in a well-marked Charcot's joint in a patient of C. K. Mills at the Philadelphia Hospital. Spontaneous fractures may occur. Among other trophic

disturbances may be mentioned atrophy of the muscles, which is usually a late manifestation, but may be localized and associated with neuritis. In any very large collection of cases many instances of atrophy are found, due either to involvement of the anterior horns or to peripheral neuritis.

Cerebral Symptoms.—Hemiplegia may develop at any stage of the disease, more commonly when it is well advanced. It may be due to hæmorrhagic softening in consequence of disease of the vessels or to progressive cortical changes. Hemianæsthesia is sometimes present. Very rarely the hemiplegia is due to coarse syphilitic disease.

Dementia paralytica frequently exists with tabes, and it may be extremely difficult to determine which has been the primary affection. In a majority of the cases the locomotor ataxia has preceded the symptoms of general paresis. In other instances melancholia, dementia, or paranoia develop.

(*c*) **Paralytic Stage.**—After persisting for an indefinite number of years the patient gradually loses the power of walking and becomes bedridden or paralyzed. In this condition he is very likely to be carried off by some intercurrent affection, such as pyelo-nephritis, pneumonia, or tuberculosis.

The Course of the Disease.—A patient may remain in the pre-ataxic stage for an indefinite period, and the loss of knee-jerk and the gray atrophy of the optic nerves may be the sole indications of the true nature of the disease. In such cases incoördination rarely develops. In a majority of cases the progress is slow, and after six or eight years, sometimes less, the ataxia is well developed. The symptoms may vary a good deal; thus the pains, which may have been excessive at first, often lessen. The disease may remain stationary for years; then exacerbations occur and it makes rapid progress. Occasionally the disease seems to be arrested. There are instances of what may be called acute ataxia, in which, within a year or even less, the incoördination is marked, and the paralytic stage may develop within a few months. The disease itself rarely causes death, and after becoming bedridden the patient may live for fifteen or twenty years.

Diagnosis.—In the pre-ataxic stage the combination of lightning pains and the absence of knee-jerk is distinctive. The association of progressive atrophy of the optic nerves with loss of knee-jerk is also characteristic. The early ocular palsies are of the greatest importance. A squint, ptosis, or the Argyll-Robertson pupil may be the first symptom, and may exist with the loss only of the knee-jerk. Loss of the knee-jerk alone, however, does occasionally occur in healthy individuals.

The diseases most likely to be confounded with locomotor ataxia are: (1) *Peripheral Neuritis.*—The pseudo-tabetic gait of arsenical, alcoholic, or diabetic paralysis is quite unlike that of locomotor ataxia. In these forms there is a paralysis of the feet and the leg is lifted high in order that the toes may clear the floor. The use of the word tabes in this con-

nection should no longer be continued. If in any doubt, the absence of the lightning pains and eye symptoms and the history will suffice in the majority of cases to make the diagnosis clear. In diphtheritic paralysis the early loss of knee-jerk and the associated eye symptoms may suggest tabes, but the history, the existence of paralysis of the throat, and the absence of pains render a diagnosis easy.

(2) *Ataxic Paraplegia.*—Marked incoördination with spastic paralysis is characteristic of the condition which Gowers has termed ataxic paraplegia. In a majority of the cases this affection is distinguished also by the absence of pains and of eye symptoms.

(3) *Cerebellar Disease.*—The cerebellar incoördination has only a superficial resemblance to that of locomotor ataxia; the knee-jerk is present, there are no lightning pains, no sensory disturbances; while, on the other hand, there are headache, optic neuritis, and vomiting.

(4) Some *acute affections* involving the posterior columns of the cord may be followed by incoördination and resemble tabes very closely. In a case recently under my care, the gait was characteristic and Romberg's symptom was present. The knee-jerk, however, was retained and there were no ocular symptoms. The condition had developed within three or four months, and there was a well-marked history of syphilis. Under large doses of iodide of potassium the ataxia and other symptoms completely disappeared.

(5) *General Paresis.*—In some cases this offers a serious difficulty. In the first place, in general paresis, tabetic symptoms often develop; on the other hand, there are cases of locomotor ataxia in which, toward the end, there are symptoms of general paresis. Cases of unusually acute ataxia with mental symptoms belong, as a rule, to the former disease. The question will be considered under general paresis.

(6) Visceral crises and neuralgic symptoms may lead to error, and in middle-aged men with severe, recurring attacks of gastralgia it is always well to bear in mind the possibility of tabes, and to make a careful examination of the eyes and of the knee-jerk.

Prognosis.—Complete recovery cannot be expected, but arrest of the progress is not uncommon and a marked amelioration of the symptoms is frequent. Optic-nerve atrophy, one of the most serious events in the disease, has this hopeful aspect—that incoördination rarely follows and the progress may be arrested. The optic atrophy itself is occasionally checked. On the whole, the prognosis in tabes is bad. The experience of such men as Weir Mitchell, Charcot, and Gowers is distinctly opposed to the belief that locomotor ataxia is ever completely cured.* No such instance has come under my personal observation.

Treatment.—To arrest the progress and to relieve, if possible, the symptoms are the objects which the practitioner should have in view. A

*For a study of the reputed cures, see L. C. Gray, N. Y. Medical Journal, Nov., 1889.

quiet, well-regulated method of life is essential. It is not well, as a rule, for a patient to give up his occupation so long as he is able to keep about and perform ordinary work. I know tabetics who have for years conducted large businesses, and there have been several notable instances in our profession of men who have risen to distinction in spite of the existence of this disease. Excesses of all sorts, more particularly *in baccho et venere*, should be carefully avoided. A man in the pre-ataxic stage should not marry.

Care should be taken in the diet, particularly if gastric crises have occurred. To secure arrest of the disease many remedies have been employed. Although syphilis plays such an important *rôle* in the etiology, it is universally acknowledged that neither mercury nor the iodide of potassium have as a rule the slightest influence over the tabetic lesions. To this there is but one exception—when the syphilis is comparatively recent; when the symptoms develop within two years of the primary infection, there is then a possibility of arrest by mercury and iodide of potassium. However, they do not always relieve. In two cases of very rapidly progressing tabes following syphilis this medication was of no avail. Not only is an anti-mercurial treatment of no benefit in the majority of cases of locomotor ataxia, but my experience tallies with that of Gowers in that it may even hasten the progress of the disease. Of remedies which may be tried and are believed by some writers to retard the progress, the following are recommended: Arsenic in full doses, nitrate of silver in quarter-grain doses, Calabar bean, ergot, and the preparations of gold.

The treatment by suspension introduced a few years ago has already been practically abandoned. Good effects certainly have followed in a few cases, but it was unreasonable from the outset, either on therapeutic or scientific grounds, to hope that by such a measure permanent changes could be induced in the pathological condition. The benefits were due in great part to suggestion and to psychical effects. In any case it must be used with caution.

For the pains, complete rest in bed, as advised by Weir Mitchell, and counter-irritation to the spine (either blisters or the thermo-cautery) may be employed. The severe spells which come on particularly after excesses of any kind are often promptly relieved by a hot bath or by a Turkish bath. A prolonged course of nitrate of silver seems in some cases to allay the pains and lessen the liability to the attacks. I have never seen ill effects from its use in the spinal scleroses. Antipyrin and antifebrin may be employed, and occasionally do good, but their analgesic powers in this disease have been greatly overrated. Cannabis indica is sometimes useful. In the severe paroxysms of pain hypodermics of morphia or of cocaine must be used. The use of morphia should be postponed as long as possible. Electricity is of very little benefit. For the severe attacks of gastralgia, morphia is also required. The laryngeal crises are rarely dangerous. An application of cocaine may be made during the spasm, or a few whiffs of chloroform may be given, or nitrite of amyl. In all cases of tabes

with increased arterial tension the prolonged use of nitroglycerin, given in increasing doses until the physiological effect is produced, is of great service in allaying the neuralgic pains and diminishing the frequency of the crises. Its use must be guarded when there is aortic insufficiency. The special indication is increased tension. The bladder symptoms demand constant care. When the organ cannot be perfectly emptied the catheter should be used, and the patient may be taught its use and how to keep it thoroughly sterilized.

III. Hereditary Ataxia (*Friedreich's Ataxia*).

In 1861 Friedreich reported six cases of a form of hereditary ataxia, and the affection has usually gone by his name. Unfortunately, *paramyoclonus multiplex* is also called Friedreich's disease; so it is best, if his name is used in connection with this affection, to term it Friedreich's ataxia. It is a very different disease in many respects from ordinary tabes. It may or may not be hereditary. It is really a family disease, several brothers and sisters being, as a rule, affected. The 143 cases analyzed by Griffith occurred in 71 unrelated families. In his series inheritance of the disease itself occurred in only 33 cases. Various influences in the parents have been noted; alcoholism in only 7 cases. Syphilis has rarely been present. Of the 143 cases, 86 were males and 57 females. The disease sets in early in life, and in Griffith's series 15 occurred before the age of two years, 39 before the sixth year, 45 between the sixth and tenth years, 20 between the eleventh and fifteenth years, 18 between the sixteenth and twentieth years, and 5 between the twentieth and twenty-fifth years.

The *morbid anatomy* shows an extensive sclerosis of the posterior and lateral columns of the spinal cord. The periphery, and the cerebellar tracts are usually involved. The recent observations of Déjérine and Letulle are of special interest, since they seem to indicate that the change in this disease is a neurogliar (ectodermal) sclerosis, differing entirely from the ordinary spinal sclerosis. According to this view, Friedreich's disease is a gliosis of the posterior columns due to developmental errors.

Symptoms.—The ataxia is unlike the ordinary form. The incoordination begins in the legs, but the gait is peculiar. It is swaying, irregular, and more like that of a drunken man. There is not the characteristic stamping gait of the true tabes. Romberg's symptom may or may not be present. The ataxia of the arms occurs early and is very marked; the movements are almost choreiform, irregular, and somewhat swaying. In making any voluntary movement the action is overdone, the prehension is claw-like, and the fingers may be spread or overextended just before grasping an object. The hand frequently moves about an object for a moment and then suddenly pounces upon it. There are irregular, swaying movements, some of which are choreiform, of the head

and shoulders. There is present in many cases what is known as static ataxia, that is to say, ataxia of quiet action—irregular, slow movements of the fingers or the hands while at rest.

Sensory symptoms are not usually present. The reflexes may be lost. In Griffith's table they were abolished in 91 cases.

Nystagmus is a characteristic symptom. Atrophy of the optic nerve rarely occurs. A striking feature is early deformity of the feet. There is talipes equinus, and the patient walks on the outer edge of the feet. The big toe is flexed dorsally on the first phalanx. Lateral curvature of the spine is very common.

Trophic lesions are rare. As the disease advances paralysis comes on and may ultimately be complete. Some of the patients never walk.

Disturbance of speech is common. It is usually slow and scanning; the expression is often dull; the mental power is, as a rule, maintained, but late in the disease becomes impaired.

The *diagnosis* of the disease is not difficult when several members of a family are affected. The onset in childhood, the curious form of incoördination, the early talipes equinus, the position of the great toe, the scoliosis, the nystagmus, and scanning speech make up an unmistakable picture. The disease is often confounded with chorea, with the ordinary form of which it has nothing in common. With hereditary chorea it has certain similarities, but usually this disease does not set in until after the thirtieth year.

The disease lasts for many years and is incurable. Care should be taken to prevent contractures.

IV. Syringo-myelia.

Definition.—A gliomatous new formation about the central canal of the spinal cord, with cavity formation.

The disease has attracted a good deal of attention within the past few years, and has a definite clinical interest since cases can now be diagnosed.

Etiology and Morbid Anatomy.—Syringo-myelia must be distinguished from dilatation of the central canal—hydromyelus—slight grades of which are not very uncommon either as a congenital condition or as a result of the pressure of tumors. The cavity of syringo-myelia has a variable extent in the cord, sometimes existing in the entire length, but in many cases involving only the cervical and dorsal regions or a more limited area. It is usually in the posterior portion of the cord and extends into one posterior cornu. The transverse section may be oval or circular or narrow and fissure-like. It varies at different levels. The condition is now regarded as a *gliosis*, a development of embryonal neurogliar tissue in which hæmorrhage or degeneration takes place with the formation of cavities.

Symptoms.—The disease, which is of slow development, makes its appearance, as a rule, about adolescence, and may persist for fifteen or twenty years. There are irregular pains, chiefly in the cervical region; muscular atrophy develops, which may be confined to the arms, or sometimes extends to the legs. The reflexes are increased and a spastic condition develops in the legs. Ultimately the clinical picture may be that of an amyotrophic lateral sclerosis. The tactile sensation is usually intact and the muscular sense is retained, but painful and thermic sensations are not recognized, or there may be in rare instances complete anæsthesia of the skin and of the mucous membranes (Déjérine). This combination of loss of painful and thermic sensations with paralysis of an amyotrophic type is regarded as pathognomonic of the disease. The special senses are usually intact and the sphincters uninvolved. Trophic troubles are not uncommon. Owing to the loss of the painful and heat sensations, the patients are apt to injure themselves. A man aged seventy, whom I saw with Déjérine at the Bicêtre, had had the symptoms for over twenty-five years. Loss of sensation had preceded the atrophy, and the terminal phalanx of the middle finger was charred, as he experienced no sensation whatever when the hot end of the cigarette neared his finger. Scoliosis also may be present in these cases. The loss of painful and thermic impressions is due to the fact that these pass to the brain in the peri-ependymal gray matter, particularly that portion in the posterior roots, which is almost constantly involved in syringo-myelia. The tactile sensation is retained because the postero-external column is uninvolved.

In typical cases the *diagnosis* is easy. The combination of an amyotrophic paralysis, the picture of progressive muscular atrophy of the Aran-Duchenne type, with retention of tactile and loss of thermic and painful sensation, is probably pathognomonic of the disease. Of affections with which it may be confounded, anæsthetic leprosy is the most important, since the anæsthesia and the wasting may closely simulate it; but, as a rule, in leprosy trophic changes are more or less marked. There is often loss of phalanges and there is no characteristic dissociation of sensory impressions.

There is a remarkable affection confined to a district of Brittany and known as Morvan's disease, after the physician who described it. The disease is chronic and characterized by neuralgic pains, cutaneous anæsthesia, and painless and destructive whitlows. In Gombault's autopsy neuritis was found, but it could not be decided, owing to the state of the cord when examined, whether cavities existed or not. Joffroy reports a case in which syringo-myelia was present and claims the affections are identical. The curious distribution of the disease and the fact that at least 20 cases have occurred in a population of 5,000, suggest that it is possibly a peripheral neuritis of infectious origin. Church, of Chicago, has reported case in which, with features believed to be characteristic of syringo-myelia, the patient had the painless and destructive whitlows which form so special a feature in Morvan's disease.

V. Compression of the Spinal Cord (*Compression Myelitis*).

Definition.—Interruption of the functions of the cord by slow compression.

Etiology.—Caries of the spine, new growths, aneurism, and parasites are the important causes of slow compression. Caries, or Pott's disease, as it is usually called, after the surgeon who first described it, is in the great majority of instances a tuberculous affection. In a few cases it is due to syphilis and occasionally to extension of disease from the pharynx. It is most common in early life, but may occur after middle age. It follows trauma in a few cases. Compression occasionally results from aneurism of the thoracic aorta or the abdominal aorta, in the neighborhood of the cœliac axis.

Malignant growths frequently cause a compression paraplegia. A retroperitoneal sarcoma or the lymphadenomatous growths of Hodgkin's disease may invade the vertebræ. More commonly, however, the involvement is secondary to scirrhus of the breast.

Of parasites, the echinococcus and the cysticercus occasionally occur in the spinal canal.

Symptoms.—These may be considered as they affect the bones, the nerves, and the cord.

(1) **Vertebral.**—In malignant disease and in aneurism erosion of the bodies may take place without producing any deformity of the spine. In caries, on the other hand, it is the rule to find more or less deformity, amounting often to angular curvature. The compression is largely due to the thickening of the dura and the presence of caseous and inflammatory products between this membrane and the bone. The compression is rarely produced directly by the bone. Pain is a constant and, in the case of aneurism and tumor, agonizing feature. In caries, the spinal processes of the affected vertebræ are tender on pressure, and pain follows jarring movements or twisting of the spine. There may be extensive tuberculous disease without much deformity, particularly in the cervical region.

(2) **Nerve-root Symptoms.**—These result from compression of the nerve-roots as they pass out between the vertebræ. It is remarkable how frequently, even in extensive caries, they escape and the patient does not complain of radiating pains in the distribution of the nerves from the affected segment. Pains are more common in cancer of the spine secondary to that of the breast, and in such cases may be agonizing. There may be acutely painful areas of hyperæsthesia of the skin or anæsthesia—the *anæsthesia dolorosa*. Trophic disturbances may occur, particularly herpes. In the cervical or lumbar regions pressure on the anterior roots may give rise to wasting of the muscles supplied by the affected nerves.

(3) **Cord Symptoms.** (*a*) *Cervical Region.*—Not infrequently the caries is high up between the axis and the atlas or between the latter and the occipital bone. In such instances a retropharyngeal abscess may be

present, giving rise to difficulty in swallowing. There may be spasm of the cervical muscles, the head may be fixed, and movements may either be impossible or cause great pain. In a case of this kind in the Montreal General Hospital movement was liable to be followed by transient, instantaneous paralysis of all four extremities, owing to compression of the cord. In one of these attacks the patient died.

In the lower cervical region there may be signs of interference with the cilio-spinal centre and dilatation of the pupils. Occasionally there is flushing of the face and ear of one side or unilateral sweating. Deformity is not so common, but healing may take place with the production of a callus of enormous breadth, and complete rigidity of the neck. The nerves of the upper extremities may be involved, and shooting pains may occur in the arm.

(*b*) *Dorsal Region.*—The deformity is here more marked and pressure symptoms are more common. The time of onset of the paralysis varies very much. It may be an early symptom, even before the curvature is manifest. More commonly it is late, occurring many months after the curvature has developed. The paraplegia is slow in its development; the patient at first feels weak in the legs or has disturbance of sensation, numbness, tingling, pins and needles. The girdle sensation may be marked, or severe pains in the course of the intercostal nerves. Motion is, as a rule, more quickly lost than sensation. Finally, there is complete interruption with the production of paraplegia, usually of the spastic type, with exaggeration of the reflexes. This may persist for months, or even for more than a year, and recovery still be possible.

(*c*) *Lumbar Region.*—In the lower dorsal and lumbar regions the symptoms are practically the same, but the sphincter centres are involved and the reflexes are not exaggerated.

Diagnosis.—Caries is by far the most frequent cause of slow compression of the cord, and when there are external signs the recognition is easy. There are cases in which the exudation in the spinal canal between the dura and the bone leads to compression before there are any signs of caries, and if the root symptoms are absent it may be extremely difficult to arrive at a diagnosis. Janeway has called attention to persistent lumbago as a symptom of importance in masked Pott's disease, particularly after injury. Brown-Séquard's paralysis is more common in tumor and in injuries than in caries. Pressure on the nerve-roots, too, is less frequent in caries than in malignant disease. The cervical form of pachymeningitis also produces a pressure paralysis, the symptoms of which have already been detailed. Pressure from cancer is naturally suggested when spinal symptoms follow within a few years after an operation. In paraplegia following tumor of the vertebra secondary to cancer of the breast, and in the erosion of the spine by retroperitoneal growths, the suffering is most intense. The condition has been well termed *paraplegia dolorosa.*

Treatment.—In compression by aneurism or tumor the condition is hopeless. In the former the pains are often not very severe, but in the latter morphia is always necessary. On the other hand, compression by caries is often successfully relieved even after the paralysis has persisted for a long period. When caries is recognized early, rest and support to the spine by the various methods now used by surgeons may do much to prevent the onset of paraplegia. When paralysis has developed, rest with extension gives the best hope of recovery. It is to be remembered that restoration may occur after compression of the cord has lasted for many months, or even more than a year. Cases have been cured by rest alone; the extradural and inflammatory products are absorbed and the caries heal. The most brilliant results in these cases have been obtained by suspension, a method introduced by J. K. Mitchell in 1826, and pursued with remarkable success by his son, Weir Mitchell. During my association with the Infirmary for Nervous Diseases I had numerous opportunities of witnessing the really remarkable effects of persistent suspension, even in apparently desperate and protracted cases. Mitchell's conclusions are that suspension should be employed early in Pott's disease; that used with care it enables us slowly to lessen the curve; that in these cases there must be, in some form, a replacement of the crumpled tissues; that unless there is great loss of power the use of the spine-car or chair of J. K. Mitchell enables suspension, especially in children, to be combined with some exercise; that no case of Pott's disease should be considered desperate without its trial; that suspension has succeeded after failures of other accepted methods; that the pull probably acts more or less directly on the cord itself, and that the gain is not explicable merely by obvious effects on the angular bony curve; that the methods of extension to be used in carious cases may be very varied, provided only we get active extension; that the plan and the length of time of extension must be made to conform to the needs, endurance, and sensation of the individual case. It may be months before there are any signs of improvement. In protracted cases, after suspension has been tried for months, laminectomy may be considered, and has in some instances been successful.*

The general treatment of caries is that of tuberculosis—fresh air, good food, cod-liver oil, and arsenic. Counter-irritation in these instances is of doubtful value.

Unilateral Lesions of the Spinal Cord (Brown-Séquard's Paralysis).—Tumors, stab wounds, and less frequently fracture or caries, may destroy one half of the cord, causing a peculiar and definite palsy, which was first recognized by Brown-Séquard, after whom it has been named. In a unilateral lesion the motor fibres are interrupted after their decussation in the medulla, consequently there is paralysis of the leg, or, if the lesion is in the cervical cord, of the arm and leg on the same side—spinal hemi-

* See full discussion of the subject by J. William White, Therapeutic Gazette, 1891.

plegia. As the sensory fibres, entering the cord through the posterior roots, decussate at once and ascend in the opposite half of the cord, there is loss of sensation on the side opposite to the lesion, so that in hemisection of the cervical cord above the brachial enlargement there is motor paralysis of the arm and leg of the same side and anæsthesia of the arm and leg of the opposite side. The anæsthesia may be only to painful and to thermic sensation. In many cases the tactile sensation is unimpaired. The muscular sense is diminished on the same side as the lesion, and on this side also the skin is hyperæsthetic, so that a slight irritation is felt very acutely. Of this phenomenon, which may persist for years, no satisfactory explanation has been given. Just above the level of the hyperæsthesia there is a narrow zone of anæsthesia, which is at the exact physiological level of the lesion and corresponds to the fibres coming from the same side, which are involved at once on entering the cord. Above this again there is a narrow zone of hyperæsthesia. The reflexes are usually increased on the side of the lesion and the temperature is slightly raised.

The following table of Gowers illustrates the distribution of these various symptoms in a hemi-lesion of the cord:

Cord.

Zone of cutaneous hyperæsthesia. Zone of cutaneous anæsthesia.	Lesion.	
Motor palsy. Hyperæsthesia of skin. Muscular sense impaired. Reflex action first lessened and then increased. Temperature raised.		Muscular power normal. Loss of sensibility of skin. Muscular sense normal. Reflex action normal. Temperature same as that above lesion.

It is only in exceptional cases that all these features are met with in a case of Brown-Séquard's paralysis, and the condition may be transitory and rapidly replaced by paraplegia.

VI. Lesions of the Cauda Equina and Conus Medullaris.

The spinal cord extends only to the second lumbar vertebra. Injury, tumors, and caries at or below this level involve not the cord itself, but the bundle of nerves known as the cauda equina and the terminal portion of the cord, the conus medullaris. Much attention has been given recently to lesions of this part. The whole subject is admirably discussed in a recent work by Thorburn.* Fractures and dislocations are common in the lumbo-sacral region, tumors not infrequently involve the filaments of the cauda equina, and some of the nerves may be entangled in the cicatrix of a spina bifida.

* A Contribution to the Surgery of the Spinal Cord. By William Thorburn. London, 1890.

In a fracture or dislocation of the first lumbar vertebra the conus medullaris may be compressed with the last sacral nerves given off from it. In a case recently reported by Kirchhoff there was laceration of the conus with complete paralysis of the bladder and rectum, a case which is held to favor the view that the ano-vesical centre in man is situated in this region of the cord. There are several instances on record in which injury of the cauda equina has produced paralysis of the bladder and rectum alone, sometimes with a slight patch of anæsthesia in the neighborhood of the coccyx or the perinæum. More commonly branches of the sacral or lumbar nerve roots are involved producing an irregularly distributed motor and sensory paralysis in the legs. When the lumbar nerve-roots from the second to the fifth are compressed there is paralysis of the muscles of the legs, with the exception of the flexors of the ankles, the peronæi, the long flexors of the toes, and the intrinsic muscles of the feet, and loss of sensation in the front, inner, and outer part of the thighs, the inner side of the legs, and the inner side of the foot. The sacral roots may alone be involved. Thus in a case which I have reported the patient fell from a bridge and had paralysis of the legs and of the bladder and rectum. When seen sixteen years after the injury, there was slight weakness, with wasting of the left leg; there was complete loss of the function in the ano-vesical and genital centres, and anæsthesia in a strip at the back part of the thigh (in the distribution of the small sciatic), and of the perinæum, scrotum, and penis. The urethra was also insensitive. In a second case, in a young man with a healed spina bifida there was, with a small area of anæsthesia, involvement of the bladder and rectum, but retention of the sexual power.

Starr's table, given in the section on motor localization, will be found useful in determining the nerve fibres and segments involved in these cases of injury of the cauda equina.

VII. Tumors of the Spinal Cord and its Membranes.

New growths may develop in the cord or in its membranes, or may extend into them from the spine. The first two alone will be considered. Occasionally lipoma and parasites occur in the extradural space. Within the dura fibromata, sarcomata, and syphilitic and tuberculous growths are most common. In the cord itself, and attached to the pia mater, the tuberculous, syphilitic, and gliomatous growths are most frequent. Of 50 cases of tumor of the spinal cord and its envelopes analyzed by Mills and Lloyd, only 3 were parasitic. Of these 26 were some form of neoplasm, of which sarcomata were most common, 5 were gummatous, and 4 tuberculous. Herter has recently reported 3 cases of solitary tubercle in the cord, and has analyzed others from the literature. Of 24 cases in which the age was given, 15 occurred between the ages of fifteen and thirty-five, and 5 before the fifth year. The tumor is most common in

the dorsal and lumbar regions, and is usually met with in connection with tuberculous lesions elsewhere.

The anatomical effects of tumors are very varied. Slow compression is usually produced by growths external to the cord, and it is remarkable what a high grade of compression the cord will bear without serious interference with its functions. In cases of prolonged interruption ascending and descending degenerations occur. Tumors developing within the cord may lead to syringo-myelia. And, lastly, tumors not infrequently excite intense myelitis.

Symptoms.—These will naturally vary a good deal with the segment involved and with the degree of pressure and the extent of implication of the nerve-roots.

Within the cord the symptoms are those of a gradually progressing paraplegia, which may at first have the picture of a Brown-Séquard paralysis. Atrophy follows the involvement of the anterior cornua, and vaso-motor disturbances may be marked. The reflexes are lost at the level of the lesion, but if in the dorsal cord, the reflexes are retained in the legs. The symptoms are apt to be complicated with those of acute or subacute myelitis, which may completely alter the clinical picture. Tumors of the spinal membranes are characterized by the early onset and persistence of the root symptoms, which consist of radiating pains, girdle sensation, hyperæsthesia, or anæsthesia in various portions of the trunk. There may even be severe pain in the anæsthetic areas. Irritation of the motor roots may cause spasm of the muscles supplied, or wasting with paralysis. The paraplegia supervenes some time after the occurrence of the root symptoms. In the dorsal region the level of the growth is usually accurately defined by the level of the pain and the condition of the reflexes.

The diagnosis of tumor within the cord is sometimes easy, the characteristic features being the constancy and severity of the root symptoms at the level of the growth and the progressive paralysis. Caries may cause identical symptoms, but the radiating pains are rarely so severe. Cervical meningitis simulates tumor very closely, and in reality produces identical effects, but the very slow progress and the bilateral character from the outset may be sufficient to distinguish this.

In chronic transverse myelitis the symptoms may, according to Gowers, simulate tumor very closely and present radiating pains, a sense of constriction, and progressive paralysis.

The nature of the tumor can rarely be indicated with precision. With a marked syphilitic history gumma may naturally be suspected, and with coexisting tuberculous disease a solitary tubercle.

Treatment.—If the possibility of syphilitic infection is present the iodide of potassium should be given in large and increasing doses. For the severe pains counter-irritation is sometimes beneficial, particularly the thermo-cautery; morphia is, however, often necessary.

In a few instances tumors of the cord or of the membranes are amena-

ble to surgical treatment. The removal by Victor Horsley of a growth from the membranes of the cord in a patient of Gowers' was one of the most brilliant operations of modern surgery.

VIII. Progressive (Spinal) Muscular Atrophy

(Chronic Degeneration of the Motor Nuclei—Poliomyelitis Anterior Chronica).

Definition.—A disease characterized by degeneration of groups of the motor nuclei in the cord and medulla, with wasting of the corresponding muscles. The pyramidal tracts are usually involved, and the paralysis may have a spastic character. In some cases the degeneration has been traced to the ganglion cells of the motor cortex.

Three affections, as a rule described apart, belong together in this category: (*a*) Progressive muscular atrophy of spinal origin; (*b*) amyotrophic lateral sclerosis; and (*c*) progressive bulbar paralysis. A slow atrophic change in the motor nuclei is the anatomical basis, and the disease, as Charcot states, is one of the whole motor path, involving, in many cases, the cortical, bulbar, and spinal centres. There may be simple muscular atrophy with little or no spasm, or progressive wasting with marked spasm and great increase in the reflexes. In others, there are added symptoms of involvement of the motor nuclei in the medulla—a glosso-labio-laryngeal paralysis; while in others, again, with atrophy (especially of the arms), a spastic condition of the legs, and bulbar phenomena, tremors develop and signs of cortical lesion. These various stages may be traced in the same case. I have for ten years had under observation a man whose illness began with weakness and atrophy of the hand muscles. Gradually the legs began to get stiff and the gait spastic; the arms subsequently wasted and the reflexes were increased. After these symptoms had persisted with increasing intensity for six or seven years, certain of the motor nuclei of the medulla became involved, the speech became thick, and the movements of the lips and tongue were impaired. Tremor has developed of late in the arms and hands. With these chronic changes the visceral functions have remained unimpaired and the mind unaffected. It has been a lesion of the motor segments, beginning in the lower and gradually extending upward. The disease began as progressive atrophy, and gradually assumed a typical picture of amyotrophic lateral sclerosis, and now the bulbar features are well marked and the tremor would indicate that the cortex is also involved.

For convenience, bulbar paralysis will be considered separately, and I shall here take up together *progressive muscular atrophy* and *amyotrophic lateral sclerosis.*

The disease is known as the Aran-Duchenne type of progressive muscular atrophy, after the French physicians who early described it, and as Cruveilhier's palsy. Lockhard Clarke demonstrated that it was a spinal

lesion. Charcot separated the two types—one with simple wasting, in which the anterior horns are alone involved; and the other in which, with degeneration of the cornua, the pyramidal tracts are affected, causing wasting *plus* a spastic condition. To this he gave the name of amyotrophic lateral sclerosis. There is but little evidence, however, to show that the anterior horns are ever affected without secondary changes in the pyramidal tracts, and Leyden and Gowers regard the two diseases as identical.

Etiology.—The cause of the disease is unknown. It is more frequent in males than in females. It attacks adults, developing after the thirtieth year, though occasionally younger persons are attacked. A large majority of all cases of progressive muscular atrophy under twenty-five years of age are of myopathic (i. e., muscular), not myelopathic (i. e., spinal) origin. Cold, wet, exposure, fright, and mental worries are mentioned as possible causes. Hereditary influences are present in certain cases. The father of the man whose case is referred to above died of progressive wasting of the muscles, but there have been no other cases in the family. It is highly probable that when many members of a family are affected the disease is not spinal, but an idiopathic muscular atrophy; and yet, in the Farr family, which I recorded a few years ago, in which thirteen members were affected in two generations, with the exception of two, the cases occurred or proved fatal above the age of forty, and the late onset speaks rather for a spinal affection. The amyotrophic form may develop late in life—after seventy—as a senile change.

Morbid Anatomy.—The following are the important anatomical changes: (*a*) The muscles waste and undergo fatty and sclerotic changes. The terminal branches of the motor nerves are degenerated. (*b*) The anterior roots are atrophied in those sections of the cord corresponding to the wasted muscles. (*c*) The gray matter shows the most marked alteration. The large ganglion cells of the anterior horns are atrophied, or, in places, have entirely disappeared, the neurogliar tissue is increased, and the fibres of the anterior nerve-root passing through the white matter are wasted. (*d*) In a majority of all the cases there is sclerosis in the antero-lateral tracts, but the direct cerebellar and the antero-lateral ascending tracts are spared. It was to this combination of atrophy of the anterior horns and sclerosis of the antero-lateral columns that Charcot gave the name amyotrophic lateral sclerosis. (*e*) The degeneration of the gray matter is rarely confined to the cord, but extends to the medulla; the motor nuclei are found extensively wasted in cases which have shown bulbar symptoms during life. (*f*) Cerebral changes also occur. The pyramidal tracts have been found degenerated through the pons and capsule, and in the motor cortex the large ganglion cells are wasted.

The essential anatomical change is a slow degeneration of the motor path, involving specially the nerve-cells of the anterior cornua and the anterior root-fibres, to which the loss of power and wasting in the muscles

are secondary. The upper segment is also involved, either simultaneously or at a later period.

Symptoms.—Irregular pains may precede the onset of the wasting. In one case the pains were about the hip and shoulder joints and the patient was treated for chronic rheumatism. The hands are first affected, and there is difficulty in performing delicate manipulations. The muscles of the ball of the thumb waste early, then the interossei and lumbricales, leaving marked depressions between the metacarpal bones. Ultimately the contraction of the flexor and extensor muscles and the extreme atrophy of the thumb muscles, the interossei, and lumbricales produces the claw-hand—*main en griffe* of Duchenne. The flexors of the forearm are usually involved before the extensors. In the shoulder-girdle the deltoid wastes first; it may waste even before the other muscles of the upper extremity. The trunk muscles are gradually attacked; the upper part of the trapezius long remains unaffected. Owing to the feebleness of the muscles which support it, the head tends to fall forward. The platysma myoides is unaffected and often hypertrophies. The arms and the trunk muscles may be much atrophied before the legs are attacked. The glutei, the vasti, and the tibialis anticus are first attacked when the disease begins in the legs. In the member of the Farr family who came under my notice (if this was really a myelopathic disorder) the wasting began in the gluteal and hamstring muscles of the left leg. The face muscles are attacked late. Ultimately the intercostal and abdominal muscles may be involved, the wasting proceeds to an extreme grade, and the patient may be actually "skin and bone," and, as "living skeletons," the cases are not uncommon in "museums" and "side-shows." Deformities and contractures result, and lordosis is almost always present. A curious twitching of the muscles (fibrillation) is a common symptom, and may occur in muscles which are not yet attacked. It is not, as was formerly supposed, a characteristic feature of the disease. The irritability of the muscle is increased. Sensation is unimpaired, but the patient may complain of numbness and coldness of the affected limbs. The galvanic and faradic irritability of the muscles progressively diminishes and may become extinct, the galvanic persisting for the longest time. In cases of rapid wasting and paralysis there may be the reaction of degeneration. The excitability of the nerve-trunks may persist after the muscles have ceased to respond. The loss of power is usually proportionate to the degree of wasting.

The foregoing description applies to the group of cases in which the atrophy and paralysis are flaccid—*atonic*, as Gowers calls it. In other cases, those which Charcot describes as amyotrophic lateral sclerosis, with the wasting there is more or less spasm, which may exist from the outset. This *tonic* atrophy may involve the legs chiefly or is present in the arms and legs. The reflexes are greatly increased. The most typical condition of spastic paraplegia may be produced. On starting to walk, the patient

seems glued to the ground and makes ineffectual attempts to lift the toes; then four or five short, quick steps are taken on the toes with the body thrown forward; and finally he starts off, sometimes with great rapidity. Some of the patients can walk up and down stairs better than on the level. The wasting is never so extreme as in the atonic form, and the loss of power may be out of proportion to it. The sphincters are unaffected. Sexual power may be lost early.

As the degeneration extends upward an important change takes place from the development of bulbar symptoms, which may, however, precede the spinal manifestations. The lips, tongue, face, pharynx, and larynx may be involved. The lips may be affected and articulation impaired for years before serious symptoms occur. In the final stage there may be tremor, the memory fails, and a condition of dementia may develop.

Gowers gives the following useful classification of the varieties of this affection: (1) Atonic atrophy, becoming extreme; (2) muscular weakness with spasm, but without wasting or with only slight wasting; and (3) atonic atrophy, rarely extreme in degree, with excess of the reflexes. These conditions may "coexist in every degree and combination—between universal atonic atrophy on the one hand and universal spastic paralysis without wasting on the other."

Diagnosis.—The affection must be distinguished from the primary muscular atrophies which usually occur in younger persons, often affect many members of a family, and have a different distribution, beginning either in the muscles of the shoulder girdle—sparing the hands or involving the face and upper-arm muscles—or the peroneal group. Muscular atrophy in the adult, beginning in the muscles of the thumbs, gradually involving the interossei and lumbricales, as a rule is of myelopathic origin.

Treatment.—The disease is incurable. I have never seen the slightest benefit from drugs or electricity. The downward progress is slow but certain, though in a few cases a temporary arrest may take place. With a history of syphilis, mercury and iodide of potassium may be tried, and Gowers recommends courses of arsenic and strychnine. Probably the most useful means is systematic massage, particularly in the spastic cases.

Bulbar Paralysis (*Glosso-labio-laryngeal Paralysis*).

An affection of the motor nuclei of the medulla oblongata, rarely primary, more commonly a part of a general degenerative affection of the nuclei of the motor path. The disease is sometimes called by the name of Duchenne. Acute and chronic forms may be recognized.

(1) *Acute bulbar paralysis* may be due to (*a*) hæmorrhagic or embolic softening in the pons and medulla; (*b*) acute inflammatory softening, analogous to polio-myelitis, occurring occasionally as a post-febrile affection.

The onset is usually sudden, hence the term apoplectiform. The cases

are almost invariably bilateral. As the nuclei presiding over the muscles of the tongue and lips are involved the speech is almost or entirely lost. The saliva drools, the lips are flabby and flaccid, swallowing may be difficult, and there may be loss of power in the laryngeal muscles. Usually these cases rapidly prove fatal, but occasionally a case with a sudden onset, like that figured by Gowers, may become chronic. In these acute cases there may be loss of power in one arm, or hemiplegia, sometimes alternate hemiplegia, with paralysis on one side of the face and loss of power on the other side of the body.

(2) *Chronic bulbar paralysis* is an affection of adult life, rarely beginning under the fortieth year, and in a great majority of the cases it is only part of a general degeneration of the motor nuclei. The disease usually begins with slight defect in the speech, and the patient has difficulty in pronouncing the dentals and linguals. The paralysis starts in the tongue, and the superior lingual muscle gradually becomes atrophied, and finally the mucous membrane is thrown into transverse folds. In the process of wasting the fibrillary tremors are seen. Owing to the loss of power in the tongue, the food is with difficulty pushed back into the pharynx. The saliva also may be increased, and is apt to accumulate in the mouth. When the lips become involved the patient can neither whistle nor pronounce the vowels *o* and *u*. The mouth looks large, the lips are prominent, and there is constant drooling. The food is masticated with difficulty. Swallowing becomes difficult, owing partly to the regurgitation into the nostrils, partly to the involvement of the pharyngeal muscles. The muscles of the vocal cords waste and the voice becomes feeble, but the laryngeal paralysis is rarely so extreme as that of the lips and tongue.

The course of the disease is slow but progressive. Death often results from an aspiration pneumonia, sometimes from choking, more rarely from involvement of the respiratory centres. The mind usually remains clear. The patient may become emotional. In a majority of the cases the disease is only part of a progressive atrophy, either simple or associated with a spastic condition. In the latter stage of amyotrophic lateral sclerosis the bulbar lesions may paralyze the lips long before the pharynx or larynx becomes affected.

The *diagnosis* of the disease is readily made, either in the acute or chronic form. The involvement of the lips and tongue is usually well marked, while that of the palate may be long deferred. A condition has been described, however, which may closely simulate bulbar paralysis. This is the so-called *pseudo-bulbar* form or bulbar palsy of cerebral origin. Bilateral disease of the motor cortex in the lower part of the ascending frontal convolution may cause paralysis of the lips and tongue and pharynx, which closely simulates a lesion of the medulla. Sometimes the symptoms appear on one side, but in many instances they develop suddenly on both sides. A bilateral lesion has usually been found, but in several instances the disease was unilateral.

Progressive bulbar paralysis is an incurable affection. Transient improvement may occur. Strychnine may be tried. Electricity is of doubtful benefit. Special care must be taken in feeding these patients, and when deglutition becomes much impaired the stomach-tube should be employed.

III. DISEASES OF THE BRAIN.

I. AFFECTIONS OF THE MENINGES.

Diseases of the Dura Mater (*Pachymeningitis*).

(*a*) **Pachymeningitis Externa.**—Hæmorrhage often occurs as a result of fracture. Inflammation of the external layer of the dura is rare. Caries of the bone, either extension from middle-ear disease or due to syphilis, is the principal cause. In the syphilitic cases there may be a great thickening of the inner table and a large collection of pus between the dura and the bone. In a remarkable case of this kind at the Montreal General Hospital the frontal lobes were so compressed by the thickened skull, and the purulent effusion between the bone and the dura, that the anterior vertical measurement of the brain was only 2·5 cm., while that of the posterior part was 8 cm.

Occasionally the pus is infiltrated between the two layers of the dura mater or may extend through and cause a dura-arachnitis.

The symptoms of external pachymeningitis are indefinite. In the syphilitic cases there may be a small sinus communicating with the exterior. Compression symptoms may occur with or without paralysis.

(*b*) **Pachymeningitis Interna.**—This occurs in three forms: (1) Pseudo-membranous, (2) purulent, and (3) hæmorrhagic. The first two are unimportant. Pseudo-membranous inflammation of the lining membrane of the dura is not usually recognized, but a most characteristic example of it came under my observation as a secondary process in pneumonia. Purulent pachymeningitis may follow an injury, but is more commonly the result of extension from inflammation of the pia. It is remarkable how rarely pus is found between the dura and arachnoid membranes.

Hæmorrhagic Pachymeningitis (*Hæmatoma of the Dura Mater*).

This remarkable condition, first described by Virchow, is very rare in general medical practice. During ten years no instance of it came under my observation at the Montreal General Hospital. On the other hand, in the post-mortem room of the Philadelphia Hospital, which received material from a large almshouse and asylum, the cases were not uncommon, and within three months I saw four characteristic examples, three of which came from the medical wards. On the other hand, the frequency

of the condition in asylum work may be gathered from the fact that Wigglesworth found 42 examples in a series of 400 unselected post-mortem examinations.

The disease is found chiefly in males and in persons over fifty years of age. It is most frequent in forms of chronic insanity and in chronic alcoholism. It has also been found in profound anæmia and other blood conditions, and is said to have followed certain of the acute fevers.

The morbid anatomy is interesting. Virchow's view that the delicate vascular membrane precedes the hæmorrhage is undoubtedly correct. Practically we see one of three conditions in these cases: (*a*) Subdural vascular membranes, often of extreme delicacy; (*b*) simple subdural hæmorrhage; (*c*) combination of the two, vascular membrane and blood-clot. Certainly the vascular membrane may exist without a trace of hæmorrhage—simply a fibrous sheet of varying thickness, permeated with large vessels, which may form beautiful arborescent tufts. On the other hand, there are instances in which the subdural hæmorrhage is found alone—in 15 out of Wigglesworth's 42 cases—but it is possible that in some of these at least the hæmorrhage may have destroyed all trace of the vascular membrane. In some cases a series of laminated clots are found, forming a layer from 3 to 5 mm. in thickness. Cysts may occur within this membrane. The source of the hæmorrhage is probably the dural vessels. Hugenin and others hold that the bleeding comes from the vessels of the pia mater, but certainly in the early stage of the condition there is no evidence of this; on the other hand, the highly vascular subdural membrane may be seen covered with the thinnest possible sheeting of clot, which has evidently come from the dura. The subdural hæmorrhage is usually associated with atrophy of the convolutions, and it is held that this is one reason why it is so common in the insane; but there must be some other factor than atrophy, or we should meet with it in phthisis and various cachectic conditions in which the cerebral wasting is as common and almost as marked as in cases of insanity.

The symptoms are indefinite, and the diagnosis cannot be made with certainty. Headache has been a prominent symptom in some cases, and when the condition exists on one side there may be hemiplegia. Extensive bilateral disease may exist without any symptoms whatever.

Diseases of the Pia Mater.

(*a*) **Acute Leptomeningitis.**—In this form the exudation is between the pia and the arachnoid membranes.

Etiology.—Acute inflammation of the pia mater occurs under the following circumstances: (1) As a result of an eruption of tubercles, most frequently in the basal meninges, forming the basilar or tuberculous meningitis which has been already considered (see tuberculosis). (2) In the epidemic cerebro-spinal fever. (3) Secondary to acute general diseases,

more particularly pneumonia, less frequently small-pox, typhoid fever, rheumatic fever, whooping cough, scarlet fever, and measles. In erysipelas meningitis may arise either by infection through the blood or by direct extension. Cases in which the inflammation passes through the bone are extremely rare; on the other hand, there are instances of extensive erysipelas of the face in which the disease travels along the nerve-roots and so reaches the meninges. In this group pneumonia is the only disease which is frequently followed by meningitis. In one hundred autopsies at the Montreal General Hospital in pneumonia, meningitis was found eight times, and I had several opportunities of seeing cases of similar character in Philadelphia. In septicæmia and pyæmia, including ulcerative endocarditis in this category, acute meningitis is not very rare. In ulcerative endocarditis it is common, as may be judged from the statistics which I collected of 209 cases, of which 25 were complicated with meningitis. No instance has fallen under my observation in connection with typhoid fever or rheumatic fever.

(4) Injury or disease of the bones of the skull, perforating wounds of the orbit, or as a sequence of abscess which is the result of injury. Under this section by far the most frequent cause is necrosis in the petrous portion of the temporal bone, which may excite either extensive inflammation of the pia mater or abscess of the brain. (5) In certain constitutional conditions, such as gout and Bright's disease. This form is usually basilar and comes on insidiously. Gout is usually mentioned as a cause of meningitis, but it must be extremely rare. Duckworth does not refer to it in his work, and the symptoms of the so-called cerebral gout can scarcely be separated from those of uræmia. On the other hand, in Bright's disease, I have met with at least three instances of well-marked meningitis, chiefly of the base.

(6) While in a great majority of all cases of basilar meningitis in children tubercles may be found, a simple *leptomeningitis infantum* must also be recognized. Cases are not very uncommon. Two occurred in debilitated children under my care at the Infants' Home in Montreal, and I saw at least two specimens of the kind at the Philadelphia Hospital. The condition may be limited to the meninges at the base, particularly at the posterior part, and to the under surface of the cerebellum. It has also been termed occlusive meningitis, owing to the fact that involving chiefly the posterior portion of the meninges about the cerebellum and medulla, the foramen of Magendie may be closed, with the result of acute, sometimes purulent hydrocephalus, as described by Gee and Barlow.* (7) Other causes mentioned are sun-stroke and excessive study, which are probably doubtful. Syphilis, which is a common cause of chronic meningitis, rarely induces the acute form.

Morbid Anatomy.—The basal or cortical meninges may be involved. In the form associated with pneumonia and ulcerative endocarditis the

* On the Cervical Opisthotonos of Infants, St. Bartholomew's Hospital Reports, 1878.

disease is bilateral and usually limited to the cortex. In extension from disease of the ear it is usually unilateral and may be accompanied with abscess or with thrombosis of the sinuses. In the non-tuberculous form in children, in the meningitis of chronic Bright's disease, and in cachectic conditions the base is usually involved. The vessels are injected, the subarachnoid fluid is increased and becomes opaque. The arachnoid is also turbid, and there may be a yellowish-white, creamy exudate, or a grayish-green purulent matter beneath the arachnoid. The interpeduncular space may be completely filled with the exudate, which extends upon the under surface of the cerebellum. In the cases secondary to pneumonia the effusion beneath the arachnoid may be very thick and purulent, completely hiding the convolutions. The ventricles also may be involved, though in these simple forms they rarely present the distention and softening which is so frequent in the tuberculous meningitis.

The *leptomeningitis infantum* may present a picture very similar to the tuberculous disease. There is exudation about the optic chiasma and in the Sylvian fissures and toward the cerebellum. In some instances we can say definitely that the condition is not tuberculous only after the most careful search in the meninges and central arteries, and when no tubercles are found in the lungs and bronchial glands. In other instances the meningitis may be limited to the posterior part of the base, about the pons, cerebellum, and fourth ventricle, and the lateral ventricles may present a most remarkable ependymitis. In a specimen recently shown to me by W. T. Howard, Jr., from a child aged three months (which had had an operation performed for imperforate anus), there was posterior basilar meningitis, the fourth ventricle was filled with pus, the walls thickened, rough, and infiltrated with pus; the lateral ventricles were enormously distended with pus, and the ependyma, which was from two to three millimetres in diameter, was softened and in a condition of purulent infiltration. A coccus and the *bacterium coli commune* were found in the pus. In a somewhat similar case at the Philadelphia Hospital the ependymitis was limited to the posterior and descending cornua, which were greatly distended and contained pus. The anterior cornua were little, if at all, affected, owing doubtless to the influence of gravity. This condition of intense purulent ependymitis is rare in the adult, but I remember to have seen an instance of it in a patient of Pepper's at the University Hospital, Philadelphia.

Symptoms.—I have already spoken at length of the clinical features of tuberculous meningitis, which is by far the most common and important form. The other varieties have a general resemblance to it, particularly those in which the base is affected. I have already, on several occasions, called attention to the fact that cortical meningitis is not to be recognized by any symptoms or set of symptoms from a condition which may be produced by the poison of many of the specific fevers. In the cases of so-called cerebral pneumonia, unless the base is involved and the

nerves affected, the disease is unrecognizable, since identical symptoms may be produced by intense engorgement of the meninges. In typhoid fever, in which meningitis is very rare, the twitchings, spasms, and retractions of the neck are almost invariably associated with cerebro-spinal congestion, not with meningitis.

A knowledge of the etiology gives a very important clew. Thus, in middle-ear disease the development of high fever, delirium, vomiting, convulsions, and retraction of the head and neck would be extremely suggestive of meningitis or abscess. Headache, which may be severe and continuous, is the most common symptom. In the fevers, particularly in pneumonia, there may be no complaint of headache. Delirium is frequently early, and is most marked when the fever is high. Convulsions are less common in simple than in tuberculous meningitis. They were not present in a single instance in the cases which I have seen in pneumonia, ulcerative endocarditis, or septicæmia. In the simple meningitis of children they may occur. Rigidity and spasm or twitchings of the muscles are more common. Stiffness and retraction of the muscles of the neck are important symptoms; but they are by no means constant, and are most frequent when the inflammation extends to the meninges of the cervical cord. Vomiting is a common symptom in the early stages, particularly in basilar meningitis. Constipation is usually present. Optic neuritis is rare in the meningitis of the cortex, but is not uncommon when the base is involved.

Important symptoms are due to lesions of the nerves at the base. Strabismus or ptosis may occur. The facial nerve may be involved, producing slight paralysis, or there may be damage to the fifth nerve, producing anæsthesia and, if the Gasserian ganglion is affected, trophic changes in the cornea. The pupils are at first contracted, subsequently dilated, and perhaps unequal.

Fever is present, moderate in grade, rarely rising above 103°. In the non-tuberculous leptomeningitis of debilitated children and in Bright's disease there may be little or no fever. The pulse may be increased in frequency at first and subsequently is slow and irregular.

Treatment.—There are no remedies which in any way control the course of acute meningitis. An ice-bag should be applied to the head and, if the subject is young and full-blooded, general or local depletion may be practised. Absolute rest and quiet should be enjoined. When disease of the ear is present, a surgeon should be early called in consultation, and if there are symptoms of meningo-encephalitis which can in any way be localized trephining should be practised. An occasional saline purge will do more to relieve the congestion than blisters and local depletion. I have no belief whatever in the efficacy of counter-irritation to the back of the neck, and to apply a blister to a patient suffering with agonizing headache in meningitis is needlessly to add to the suffering. If counter-irritation is deemed essential, the thermo-cau-

tery, lightly applied, is more satisfactory, because the pain inflicted is transient.

The gastro-intestinal symptoms should receive appropriate treatment. Gowers states that in two instances of septic meningitis which recovered the good effects seemed to be due to large doses of the perchloride of iron. Iodide of potassium and mercury are recommended by some authors.

The application of an ice-cap, attention to the bowels and stomach, and keeping the fever at a moderate height by sponging, are the necessary measures in a disease recognized as almost invariably fatal, and in which the cases of recovery are extremely doubtful.

(*b*) *Chronic Leptomeningitis.*—This is rarely seen apart from syphilis or tuberculosis, in which the meningitis is associated with the growth of the granulomata in the meninges and about the vessels. The symptoms in such cases are extremely variable, depending entirely upon the situation of the growth. They may closely resemble those of tumor and be associated with localized convulsions. The leptomeningitis infantum may be chronic. In the cases reported by Gee and Barlow the duration in some instances extended even to a year and a half. The involvement of the posterior part of the meninges and of the ventricles may lead to dilatation and hydrocephalus. The symptoms upon which these authors lay stress are convulsions, and retraction of the head, which is particularly marked when the child is made to sit up. There may be rigidity of the limbs and epileptiform convulsions.

II. AFFECTIONS OF THE BLOOD-VESSELS.

Hyperæmia.

Congestion of the brain has played an important part in cerebral pathology. Undoubtedly there are great variations in the amount of blood in the cerebral vessels; this is universally conceded, but how far these changes are associated with a definite group of symptoms is not quite so clear. The hyperæmia may be either active or passive.

Active hyperæmia is associated with febrile conditions, with increased action of the heart, chilling of the surface, contraction of the superficial vessels, and with the suppression of certain customary discharges. Among other recognized causes are plethora, functional irritation, such as is associated with excessive brain work, and the action of certain substances, such as alcohol and nitrite of amyl.

Passive hyperæmia results from obstruction in the cerebral sinuses and veins, engorgement in the lesser circulation, as in mitral stenosis, emphysema, from pressure on the superior cava by aneurisms and tumors, and in the venous engorgement which takes place in prolonged straining

efforts. In its most intense form it is seen in the compression of the superior cava by tumors and in death from strangulation.

The anatomical changes in congestion of the brain are by no means striking. Active hyperæmia is never visible post mortem. The veins of the cortex are distended, the gray matter has a deeper color, and its vessels are full. The arteries at the base and in the Sylvian fissures contain blood. Nothing, however, can be more uncertain or indefinite than the post-mortem appearances of hyperæmia of the brain. The most intense distention of the vessels is seen in early death during the specific fevers, or in the secondary passive congestion due to obstruction in the superior cava or in the lesser circulation.

Symptoms.—There are no characteristic or constant features of cerebral hyperæmia. It may exist in the most extreme grade without the slightest disturbance of the cerebral functions, as is witnessed frequently in the pressure of tumors on the superior vena cava. How far the headache and delirium of the early stage of the infectious fevers is to be assigned to hyperæmia of the blood-vessels of the brain it is not easy to determine. The headache, dizziness, and unpleasant sensations [illegible] insufficiency and in some instances of hypertrophy of the heart may be due to the cerebral congestion.

As a separate clinical entity, congestion of the brain rarely comes under observation. I have no knowledge of instances associated with delirium, fever, insomnia, and convulsions, or of the so-called apoplectiform variety described by some writers. Very plethoric persons are subject to attacks of headache with flushing of the face and irritability of temper, attacks which may recur frequently and are sometimes relieved by bleeding at the nose. These are usually attributed to congestion of the brain. When passive hyperæmia reaches a high grade, there may be torpor, dulness of the intellect, and ultimately deep coma.

Anæmia.

This may be induced by loss of blood, either quickly, as in hæmorrhage, or gradually, as in the severe primary and secondary anæmias. The anæmia may be local and due to causes which interfere with the blood supply to the brain, as narrowing of the vessels by endarteritis, pressure, narrowing of the aortic orifice, or it may follow an unequal distribution of the blood in consequence of dilatation of certain vascular territories. Thus, rapid distention of the intestinal vessels, such as occurs after the removal of ascitic fluid, may cause sudden death from cerebral anæmia. The commonest illustration of this is the fainting fit from emotion, in which the blood supply to the brain is insufficient on account of the diminished arterial pressure. Anæmia of the cerebral vessels may be caused by pressure of fluid in the ventricles. The partial anæmia results from obliteration of branches of the circle of Willis by embolism or throm-

bosis. Ligature of one carotid sometimes causes a transient marked anæmia and disturbance of function on one side of the brain.

The anatomical condition of the brain in anæmia is very striking. The membranes are pale, only the large veins are full, the small vessels over the gyri are empty, and an unusual amount of cerebro-spinal fluid is present. On section both the gray and white matter look extremely pale and the cut surface is moist. Very few *puncta vasculosa* are seen.

Symptoms.—The effects of anæmia of the brain are well illustrated by a fainting fit in which loss of consciousness follows the heart weakness. When the result of hæmorrhage, there are drowsiness, giddiness, inability to stand, flashes of light, and noises in the ear; the respiration becomes hurried; the skin is cool and covered with sweat; and gradually, if the bleeding continues, consciousness is lost and death may occur with convulsions. In ordinary syncope the loss of consciousness is usually transient and the recumbent posture alone may suffice to restore the patient to consciousness. In the more chronic forms of brain anæmia, such as result from the gradual impoverishment of the blood, as in protracted illness or in starvation, the condition known as irritable weakness results. Mental effort is difficult, the slightest irritation is followed by undue excitement, the patient complains of giddiness and noises in the ears, or there may be hallucinations or delirium. These symptoms are met with in an extreme grade as a result of prolonged starvation.

An interesting set of symptoms, to which the term *hydrocephaloid* was applied by Marshall Hall, occurs in the debility produced by prolonged diarrhœa in children. The child is in a semi-comatose condition with the eyes open, the pupils contracted, and the fontanelle depressed. In the earlier period there may be convulsions. The coma may gradually deepen, the pupils become dilated, and there may be strabismus and even retraction of the head, symptoms which closely simulate basilar meningitis.

Œdema of the Brain.

In the pathology of brain lesions œdema formerly played a *rôle* almost equal in importance to congestion. It occurs under the following conditions: In general atrophy of the convolutions, in which case the œdema is represented by an increase in the cerebro-spinal fluid and in that of the meshes of the pia. In extreme hyperæmia from obstruction, as in mitral stenosis or in tumors, there may be a condition of congestive œdema, in which, in addition to great filling of the blood-vessels, the substance of the brain itself is unusually moist. The most acute œdema is a local process found around tumors and abscesses. An intense infiltration, local or general, may occur in Bright's disease, and to it, as Traube suggested, certain of the uræmic symptoms may be due.

The *anatomical changes* are not unlike those of anæmia. When a sequence of progressive atrophy, the fluid is chiefly within and beneath

the membranes. The brain substance is anæmic and moist, and has a wet, glistening appearance, which is very characteristic. In some instances the œdema is more intense and local and the brain substance may look infiltrated with fluid. The amount of fluid in the ventricles is usually increased.

The *symptoms* are in great part those of anæmia, and are not well defined. As just stated, some of the cerebral features of uræmia may depend upon it. Of late years cases have been reported by Raymond, Tenneson, and Dercum, in which unilateral convulsions or paralysis have occurred in connection with chronic Bright's disease, and in which the condition appeared to be associated with œdema of the brain. The older writers laid great stress upon an apoplexia serosa, which may really have been a general œdema of the brain.

Cerebral Hæmorrhage.

The bleeding may come from branches of either of the two great groups of cerebral vessels—the *basal*, comprising the circle of Willis and the central arteries passing from it, or the *cortical group*, the anterior, middle, and the posterior cerebral vessels. In a majority of the cases the hæmorrhage is from the central branches, more particularly from those given off by the middle cerebral arteries in the anterior perforated spaces, and which supply the corpora striata and internal capsules. One of the largest of these branches which passes to the third division of the lenticular nucleus and to the hinder part of the internal capsule is so frequently involved in hæmorrhage that it has been called by Charcot *the artery of cerebral hæmorrhage.* The bleeding may be into the substance of the brain, to which alone the term cerebral apoplexy is applied, or into the membranes, in which case it is termed meningeal hæmorrhage; both, however, are usually included under the terms intracranial or cerebral hæmorrhage.

Etiology.—The conditions which produce lesions of the blood-vessels play a very important part; thus the natural tendency to degeneration of the vessels in advanced life makes apoplexy much more common after the fiftieth year. It may, however, occur in children under ten. On account of the greater liability to arterial disease (associated probably with muscular exertion and the abuse of alcohol), men are more subject to cerebral hæmorrhage than women. Heredity was formerly thought to be an important factor in this affection, and the apoplectic *habitus* or build is still referred to. By this is meant a stout, plethoric body of medium size, with a short neck. Heredity influences cerebral hæmorrhage entirely through the arteries, and there are families in which they degenerate early, usually in association with renal changes. The secondary hypertrophy of the heart brings with it serious dangers, which have already been discussed in the section upon arteries. The three special factors in

inducing arterio-sclerosis—the abuse of alcohol, syphilis, and prolonged muscular exertion—are found to be important antecedents in a large number of cases of cerebral hæmorrhage.

The endocarditis of rheumatism and other fevers may indirectly lead to apoplexy by causing embolism and aneurism of the vessels of the brain. Cerebral hæmorrhage occurs occasionally in the specific fevers and in profound alterations of the blood, as in leukæmia and pernicious anæmia. The actual exciting cause of the hæmorrhage is not evident in the majority of cases. The attack may be sudden and without any preliminary symptoms. In other instances violent exertion, particularly straining efforts or, the excited action of the heart in emotion may cause a rupture.

Morbid Anatomy.—The lesions causing apoplexy are almost invariably in the cerebral arteries, in which the following changes may lead directly to it:

(*a*) Periarteritis with the production of miliary aneurisms, rupture of which is the most common cause of cerebral hæmorrhage. They occur most frequently on the central arteries, but also on the smaller branches of the cortical vessels. On section of the brain substance they may be seen as localized, small dark bodies about the size of a pin's head. Sometimes they are seen in numbers upon the arteries carefully withdrawn from the anterior perforated spaces. According to Charcot and Bouchard, who have described them, they are most frequent in the central ganglia. In apoplexy after the fortieth year if sought for they are rarely missed.

(*b*) Aneurism of the branches of the circle of Willis. These are by no means uncommon, and will be considered subsequently.

(*c*) Endarteritis and periarteritis in the cerebral vessels most commonly lead to apoplexy by the production of aneurisms, either miliary or coarse. There are instances in which the most careful search fails to reveal anything but diffuse degeneration of the cerebral vessels, particularly of the smaller branches; so that we must conclude that spontaneous rupture may occur without the previous formation of aneurism.

The hæmorrhage may be meningeal, cerebral, or intraventricular.

Meningeal Hæmorrhage may be outside the dura, between this membrane and the bone, or between the dura and arachnoid, or between the arachnoid and the pia mater. The following are the chief causes of this form of hæmorrhage: Fracture of the skull, in which case the blood usually comes from the lacerated meningeal vessels, sometimes from the torn sinuses. In these cases the blood is usually outside the dura or between it and the arachnoid. The next most frequent cause is rupture of aneurisms on the larger cerebral vessels. The blood is usually subarachnoid. An intracerebral hæmorrhage may burst into the meninges. A special form of meningeal hæmorrhage is found in the new-born, associated with injury during birth. And lastly, meningeal hæmorrhage may occur in the constitutional diseases and fevers. The blood may be in a large quantity at

the base; in cases of ruptured aneurism, particularly, it may extend into the cord or upon the cortex. Owing to the greater frequency of the aneurisms in the middle cerebral vessels, the Sylvian fissures are often distended with blood.

Intracerebral hæmorrhage is most frequent in the neighborhood of the corpus striatum, particularly toward the outer section of the lenticular nucleus. The hæmorrhage may be small and limited to the lenticular body and the internal capsule, or it may break the centrum ovale, or burst into the lateral ventricle, or extend to the insula. Hæmorrhages confined to the white matter—the centrum ovale—are rare. Localized bleeding may occur in the crura or in the pons. Hæmorrhage into the cerebellum is not uncommon, and usually comes from the superior cerebellar artery. The extravasation may be limited to the substance or rupture into the fourth ventricle. Twice I have known sudden death in girls under twenty-five to be due to cerebellar hæmorrhage.

Ventricular Hæmorrhage.—This rarely comes from the vessels of the plexuses or of the walls. It is not infrequent in early life and may occur during birth. Of 94 cases collected by Edward Sanders, 7 occurred during the first year, and 14 under the twentieth year. In the cases which I have seen in adults it has almost always been caused by rupture of a hæmorrhage in the neighborhood of the caudate nucleus. The blood may be found in one ventricle only, but more commonly it is in both lateral ventricles, and may pass into the third ventricle and through the aqueduct of Sylvius into the fourth ventricle, forming a complete mould in blood of the ventricular system.

Subsequent Changes.—The blood gradually changes in color, and ultimately the hæmoglobin is converted into the reddish-brown hæmatoidin. Inflammation occurs about the apoplectic area, limiting and confining it, and ultimately a definite wall may be produced, inclosing a cyst with fluid contents. In other instances a cyst is not formed, but the connective-tissue proliferates and leaves a pigmented scar. In meningeal hæmorrhage the effused blood may be gradually absorbed and leave only a staining of the membranes. In other cases, particularly in infants, when the effusion is cortical and abundant, there may be localized wasting of the convolutions and the production of a cyst in the meninges. Possibly certain of the cases of porencephaly are caused in this way.

Secondary degeneration follows when the motor cortex or motor path is involved. Thus, in persons dying some years after a cerebral apoplexy which has produced hemiplegia, the degeneration may be traced in the crus, in the anterior part of the pons, in the pyramidal fibres of the medulla, in the direct fibres of the cord of the same side, and in the crossed pyramidal fibres of the opposite side (Fig. 3).

Symptoms.—These may be divided into primary, or those connected with the onset, and secondary, or those which develop later after the early manifestations have passed away.

Primary Symptoms.—Premonitory indications are rare. As a rule, the patient is seized while in full health or about the performance of some every-day action, occasionally an action requiring strain or extra exertion. Now and then instances are found in which there are sensations of numbness or tingling or pains in the limbs, or even choreiform movements in the muscles of the opposite side, the so-called prehemiplegic chorea. The onset of the apoplexy, as cerebral hæmorrhage is usually called, varies greatly. There may be sudden loss of consciousness and complete relaxation of the extremities. In such instances the name *apoplectic stroke* is particularly appropriate. In other cases the onset is more gradual and the loss of consciousness may not occur for a few minutes after the patient has fallen, or after the paralysis of the limbs is manifest. In the apoplectic attack the condition is as follows: There is deep unconsciousness; the patient cannot be roused. The face is injected, sometimes cyanotic, or of an ashen-gray hue. The pupils vary; usually they are dilated and inactive. The respirations are slow, noisy, and accompanied with stertor. Sometimes the Cheyne-Stokes rhythm may be present. The pulse is usually full, slow, and of increased tension. The temperature may be normal, but is often found subnormal, and, as in a case reported by Bastian, may sink below 95°. In cases of basal hæmorrhage the temperature, on the other hand, may be high. The urine and fæces are usually passed involuntarily. Convulsions are not common. It may be difficult to decide whether the condition is apoplexy associated with hemiplegia or sudden coma from other causes. An indication of hemiplegia may be discovered in the difference in the tonus of the muscles on the two sides. If the arm or the leg is lifted, it drops "dead" on the affected side, while on the other it falls more slowly. Rigidity also may be present. In watching the movements of the facial muscles in the stertorous respiration it will be seen that on the paralyzed side the relaxation permits the cheek to be blown out in a more marked manner. The head and eyes may be turned strongly to one side—conjugate deviation.

In other cases, in which the onset is not so abrupt, the patient may not lose consciousness, but in the course of a few hours there is loss of power, unconsciousness gradually develops, and deepens into profound coma. This is sometimes termed ingravescent apoplexy. The attack may occur during sleep. The patient may be found unconscious, or wakes to find that the power is lost on one side. Small hæmorrhages may cause hemiplegia without loss of consciousness, more particularly when they are in the territory of the central arteries.

Usually within forty-eight hours after the onset of an attack there is febrile reaction, and more or less constitutional disturbance associated with inflammatory changes about the hæmorrhage. The patient may die in this reaction, or, if consciousness has been regained, there may be delirium or recurrence of the coma. At this period the so-called early rigidity may develop in the paralyzed limbs. Trophic changes may occur,

such as sloughing or the formation of vesicles. The most serious of these is the sloughing eschar of the lower part of the back, or on the paralyzed side, which may appear within forty-eight hours of the onset and is usually of grave significance. The congestion at the bases of the lungs so common in apoplexy is regarded by some as a trophic change.

Conjugate Deviation.—In a right hemiplegia the eyes and head may be turned to the left side; that is to say, the eyes look toward the cerebral lesion. This is almost the rule in conjugate deviation of the head and eyes which occurs early in hemiplegia. When, however, convulsions or spasm develop or the state of so-called early rigidity in hemiplegia, the conjugate deviation of the head and eyes may be in the opposite direction; that is to say, the eyes look away from the lesion and the head is rotated toward the convulsed side. This symptom may be associated with cortical lesions, particularly, according to some authors, when in the neighborhood of the supramarginal and angular gyri. It may also occur in a lesion of the internal capsule or in the pons, but in the latter situation the conjugate deviation is the reverse of that which occurs in other cases, as the patient looks away from the lesion, and in spasm or convulsion looks toward the lesion. In cases in which consciousness is restored and the patient improves, the unilateral paralysis which persists is known as

Hemiplegia.—Hemiplegia is complete when it involves face, arm, and leg, or partial when it involves only one or other of these parts. This may be the result of a lesion (*a*) of the motor cortex; (*b*) of the pyramidal fibres in corona radiata and in the internal capsule; (*c*) of a lesion in the crus cerebri; or (*d*) in the pons Varolii (see Fig. 3, *x*, *y*, *z*). Hæmorrhage is perhaps the most common cause, but tumors and spots of softening may also induce it. The special details of the hemiplegia may here be considered. The face is involved on the same side as the arm and leg. This results from the fact that the facial muscles stand in precisely the same relation to the cortical centres as those of the arm and leg, the fibres of the upper motor segment of the facial nerve from the cortex decussating just as do those of the nerves of the limbs. The facial paralysis is partial, involving only the lower portion of the nerve, so that the orbicularis oculi and the frontalis muscles are uninvolved. The signs of the facial paralysis are usually well marked. There may be a slight difficulty in elevating the eyebrows or in closing the eye on the paralyzed side. The hypoglossal nerve also is involved. In consquence, the patient cannot put out the tongue straight, but it deviates toward the paralyzed side, inasmuch as the genio-hyo-glossus of the sound side is unopposed. When the hemiplegia is on the right side there may be aphasia.

The arm is, as a rule, more completely paralyzed than the leg. The loss of power may be absolute or partial. In severe cases it is at first complete. In others, when the paralysis in the face and arm is complete that of the leg is only partial. The face and arm may alone be par-

alyzed, while the leg escapes. Less commonly the leg is more affected than the arm, and the face may be only slightly involved.

Certain muscles escape in hemiplegia, particularly those associated in symmetrical movements, as the thoracic and abdominal muscles, a fact which Broadbent explains by supposing that as the spinal nuclei controlling these movements on both sides constantly act together, they may, by means of this intimate connection, be stimulated by impulses coming from only one side of the brain.

Crossed hemiplegia occurs when a lesion is in the lower section of the pons Varolii (Fig. 3, *z*), in which the facial nerve is involved as it passes through the pons after it has left its nucleus; whereas, the motor fibres involved in the lesion are above the point of their decussation, so that facial paralysis occurs on the same side as the lesion, and paralysis of the arm and leg on the opposite side.

The sensory disturbances are variable. Hemianæsthesia may coexist with hemiplegia, but in many instances there is only slight numbness of sensation. When the hemianæsthesia is marked, it is usually the result of a lesion in the internal capsule. In C. L. Dana's study of sensory localization he found that anæsthesia of organic cortical origin was always limited or more pronounced in certain parts, as the face, arm, or leg, and was generally incomplete. Total anæsthesia was either of functional or subcortical origin. Marked anæsthesia was much more common in softening than in hæmorrhage. Complete hemianæsthesia is certainly rare in hæmorrhage.

Disturbance of the special senses is not common. Hemianopia may exist on the same side as the lesion, and there may be diminution in the acuteness of the senses of hearing, taste, and smell.

As a rule, there is no wasting of the paralyzed limbs. The deep reflexes are increased on the paralyzed side, and ankle clonus may be present. The plantar and other superficial reflexes are usually diminished. The sphincters are not affected.

The *course* of the disease depends upon the situation and extent of the lesion. If slight, the hemiplegia may disappear completely within a few days or a few weeks. In severe cases the rule is that the leg gradually recovers before the arm, and the muscles of the shoulder girdle and upper arm before those of the forearm and hand. The face may recover quickly.

Except in the very slight lesions, in which the hemiplegia is transient, changes take place which may be grouped as

Secondary Symptoms.—These correspond to the chronic stage. In a case in which little or no improvement takes place within eight or ten weeks, it will be found that the paralyzed limbs undergo certain changes. The leg, as a rule, recovers enough power to enable the patient to get about, although the foot is dragged. In both arm and leg the condition of *secondary contraction* or *late rigidity* comes on and is always most marked in the upper extremity. The arm becomes permanently flexed at

the elbow and resists all attempts at extension. The wrist is flexed upon the forearm and the fingers upon the hand. The position of the arm and hand is very characteristic. There is frequently, as the contractures develop, a great deal of pain. In the leg the contracture is rarely so extreme. The loss of power is most marked in the muscles of the foot, and to prevent the toes from dragging the knee in walking is much flexed, or more commonly the foot is swung round in a half-circle.

The reflexes are at this stage greatly increased. These contractures are permanent and incurable, and are associated with a secondary descending sclerosis of the motor path. There are instances, however, in which rigidity and contracture do not occur, but the arm remains flaccid, the leg having regained its power. This *hémiplégie flasque* of Bouchard is found most commonly in children. Among other secondary changes in late hemiplegia may be mentioned the following: Tremor of the affected limbs, post-paralytic chorea, the mobile spasm known as athetosis, arthropathies in the joints of the affected side, and muscular atrophy. Athetosis and post-hemiplegic chorea will be considered in the hemiplegia of children. A word may here be said upon the subject of muscular atrophy of cerebral origin.

As a rule, atrophy is not a marked feature in hemiplegia, but in some instances it does develop. It has been shown to be due in some cases to secondary alterations in the gray matter of the anterior horns, as in a case reported by Charcot. Recently, however, attention has been called by Quincke to the fact that atrophy may follow as a direct result of the cerebral lesion. In his case, atrophy of the arm followed the development of a glioma in the anterior central convolutions. The gray matter of the anterior horns was normal. This wasting of cerebral origin occurs most frequently in children.

Diagnosis.—There are three groups of cases which offer increasing difficulty in recognition.

(1) Cases in which the onset is gradual, a day or two elapsing before the paralysis is fully developed and consciousness completely lost, are readily recognized, though it may be difficult to determine whether the lesion is due to thrombosis or to hæmorrhage.

(2) In the sudden apoplectic stroke in which the patient rapidly loses consciousness, the difficulty in diagnosis may be still greater, particularly if the patient is in deep coma when first seen.

The first point to be decided is the existence of hemiplegia. This may be difficult, although, as a rule, even in deep coma the limbs on the paralyzed side are more flaccid and drop instantly when lifted; whereas, on the non-paralyzed side the muscles retain some degree of tonus. The reflexes may be increased on the affected side and there may be conjugate deviation of the head and eyes. Rigidity in the limbs of one side is in favor of a hemiplegic lesion. It is practically impossible in a majority of

these cases to say whether the lesion is due to hæmorrhage, embolism, or thrombosis.

(3) Large hæmorrhage into the ventricles or into the pons may produce sudden loss of consciousness with complete relaxation, so that the condition may simulate coma from uræmia, alcoholism, opium poisoning, or epilepsy. The previous history and the mode of onset may give valuable information. In epilepsy convulsions have preceded the coma; in alcoholism there is a history of constant drinking, while in opium poisoning the coma develops more gradually; but in many instances the difficulty is practically very great, and on more than one occasion I have seen mortifying post-mortem disclosures under these circumstances. In ventricular hæmorrhage the coma is sudden and develops rapidly. The hemiplegic symptoms may be transient, quickly giving place to complete relaxation. Convulsions occur in many cases, and may be the very symptom to lead astray—as in a case of ventricular hæmorrhage which occurred in a puerperal patient, in whom, naturally enough, the condition was thought to be uræmic. Rigidity is often present. In hæmorrhage into the pons convulsions are frequent. The pupils may be strongly contracted, conjugate deviation may occur, and the temperature is apt to rise rapidly. The contraction of the pupils in pontine hæmorrhage naturally suggests opium poisoning. The difference in temperature in the two conditions is a valuable diagnostic point.

It may be impossible at first to give a definite diagnosis. In admissions to hospitals or in emergency cases the physician should be particularly careful about the following points: The examination of the head for injury or fracture; the urine should be tested for albumen and examined for sugar; a careful examination should be made of the limbs with reference to their degree of relaxation or the presence of rigidity, and the condition of the reflexes; the state of the pupils should be noted and the temperature taken. The most serious mistakes are made in the case of patients who are drunk at the time of the attack, a combination by no means uncommon in the class of patients admitted to hospital. Under these circumstances the case may be looked upon as one of alcoholic coma. It is best to regard each case as serious and to bear in mind that this is a condition in which, above all others, mistakes are common.

Prognosis.—From cortical hæmorrhage, unless very extensive, the recovery may be complete without a trace of contracture. This is more common when the hæmorrhage follows injury than when it results from disease of the arteries. Infantile meningeal hæmorrhage, on the other hand, is a condition which may produce idiocy or spastic diplegia.

Large hæmorrhages into the corona radiata and those which rupture into the ventricles rapidly prove fatal.

The hemiplegia which follows lesions of the internal capsule, the result of rupture of the artery of the corpus striatum, is usually persistent and followed by contracture. When the posterior fibres are involved

there may be hemianæsthesia, and later hemichorea or athetosis. In any case of cerebral apoplexy the following symptoms are of grave omen: persistence or deepening of the coma during the second and third day; rapid rise in temperature within the first forty-eight hours after the initial fall. In the reaction which takes place on the second or third day, the temperature usually rises, and its gradual fall on the third or fourth day with return of consciousness is a favorable indication. The rapid formation of bed-sores, particularly the malignant decubitus of Charcot, is a fatal indication. The occurrence of albumen and sugar, if abundant, in the urine is an unfavorable symptom.

When consciousness returns and the patient is improving, the question is anxiously asked as to the paralysis. The extent of this cannot be determined for some weeks. With slight lesions it may pass off entirely. If persistent at the end of a month some grade of permanent palsy is certain to remain, and gradually the late rigidity supervenes.

Embolism and Thrombosis (*Cerebral Softening*).

(*a*) **Embolism.**—The embolus usually enters the carotid, rarely the vertebral artery. In the great majority of cases it comes from the left heart and is either a vegetation of a fresh endocarditis or, more commonly, of a recurring endocarditis, or from the segments involved in an ulcerative process. Less often the embolus is a portion of a clot which has formed in the auricular appendix. Portions of clot from an aneurism, thrombi from atheroma of the aorta, or from the territory of the pulmonary veins, may also cause blocking of the branches of the circle of Willis. In the puerperal condition cerebral embolism is not infrequent. It may occur in women with heart-disease, but in other instances the heart is uninvolved, and the condition has been thought to be associated with the development of heart-clots, owing to increased coagulability of the blood. A majority of cases of embolism occur in chronic heart-disease. Cases are rare in the acute endocarditis of rheumatism, chorea, and febrile conditions. It is much more common in the secondary recurring endocarditis which attacks old sclerotic valves. The embolus most frequently passes to the left middle cerebral artery, as it enters the left carotid oftener than the right because of the more direct course of the blood in the former. The posterior cerebral and the vertebral are less often affected. A large plug may lodge at the bifurcation of the basilar. Embolism of the cerebellar vessels is rare.

Embolism occurs more frequently in women, owing, no doubt, to the greater frequency of mitral stenosis. Contrary to this general statement, Newton Pitt's statistics of 79 cases at Guy's Hospital indicate, however, that males are more frequently affected; for in this series there were 44 males and 35 females.

(*b*) **Thrombosis.**—Clotting of blood in the cerebral vessels occurs about

an embolus, as the result of a lesion of the arterial wall (either endarteritis with or without atheroma or, particularly, the syphilitic arteritis), in aneurisms both coarse and miliary, and very rarely as a result of abnormal conditions of the blood. Thrombosis of the cerebral vessels occasionally follows ligation of the carotid artery. The thrombosis is most common in the middle cerebral and in the basilar arteries.

Anatomical Changes following Thrombosis and Embolism.—Degeneration and softening of the territory supplied by the vessels is the ultimate result of the arterial obstruction. Blocking in a terminal artery may be followed by a condition resembling infarct, in which the territory is deeply infiltrated with blood. More commonly the change is much less striking, and the affected region may look only a little paler than normal or slightly softer. Gradually the process of softening proceeds, the tissue is infiltrated with serum and is moist, the nerve-fibres degenerate and become fatty. The neuroglia is swollen and œdematous. The color of the softened area depends upon the amount of blood. The hæmoglobin undergoes gradual transformation, and the early red color may give place to yellow. Formerly much stress was laid upon the difference between *red*, *yellow*, and *white* softening. The red and yellow are seen chiefly on the cortex. Sometimes the red softening is particularly marked in cases of embolism and in the neighborhood of tumors. The gray matter shows many punctiform hæmorrhages—capillary apoplexy. There is a variety of yellow softening—the *plaques jaunes*—common in elderly persons, which occurs in the gray matter of the convolutions. The spots are from one to two centimetres in diameter, the edges cleanly cut, and the softened area is represented by either a turbid, yellow material, or in some instances there is a space crossed by fine trabeculæ, in the meshes of which there is fluid. They result from fatty degeneration of the peripheral cortical arteries; less often the hyaline change is present. White softening occurs most frequently in the white matter, and is seen best about tumors and abscesses. Inflammatory changes are common in and about the softened areas. When the embolus is derived from an infected focus, as in ulcerative endocarditis, suppuration may follow. The final changes vary very much. The degenerated and dead tissue elements are gradually but slowly removed, and if the region is small may be replaced by growth of connective tissue and the formation of a scar. In larger regions the resorption results in the formation of a cyst, which may be crossed by connective-tissue trabeculæ. It is surprising for how long an area of softening may persist without much change.

The position and extent of the softening depend upon the obstructed artery. An embolus which blocks the middle cerebral at its origin involves both the arteries in the anterior perforated space and the cortical branches, and in such a case there is softening in the neighborhood of the corpus striatum, as well as in part of the region supplied by the cortical vessels. The freedom of anastomosis between these branches varies

a good deal. Thus, there are instances of embolism of the middle cerebral artery in which the softening has only involved the territory of the central branches, in which case blood has reached the cortex through the anterior and posterior cerebrals. When the middle cerebral is blocked (as is perhaps oftenest the case) beyond the point of origin of the central arteries, one or other of its branches is usually most involved. The embolus may lodge in the vessel passing to the third frontal convolution, or in the artery of the ascending frontal or ascending parietal; or it may lodge in the branch passing to the supramarginal and angular gyri, or it may enter the lowest branch which is distributed to the upper convolutions of the temporo-sphenoidal lobe. These are practically terminal arteries, and instances frequently occur of softening limited to a part, at any rate, of the territory supplied by them. Some of the most accurate focalizing lesions are in this way produced.

Symptoms.—Extensive thrombotic softening may exist without any symptoms. It is not uncommon in the post-mortem examination of the bodies of elderly persons to find the *plaques jaunes* scattered over the convolutions. So, too, softening may take place in the "silent" regions, as they are termed, without exciting any symptoms. When the central or cortical branches of the middle cerebral arteries are involved the symptoms are similar to those of hæmorrhage. Permanent or transient hemiplegia results. When the central arteries are involved the softening in the internal capsule is commonly followed by permanent hemiplegia. There are certain peculiarities associated with embolism and with thrombosis respectively.

In *embolism* the patient is usually the subject of heart-trouble, or there exist some of the conditions already mentioned. The onset is sudden, without premonitory symptoms. When the embolism blocks the left middle cerebral artery the hemiplegia is usually associated with aphasia. In *thrombosis*, on the other hand, the onset is more gradual; the patient has previously complained of headache, vertigo, tingling in the fingers; the speech may have been embarrassed for some days; the patient has had loss of memory or is incoherent, or paralysis begins at one part, as the hand, and extends slowly, and the hemiplegia may be incomplete or variable. Abrupt loss of consciousness is much less common, and when the lesion is small consciousness is retained. Thus, in thrombosis due to syphilitic disease, the hemiplegia may come on gradually without the slightest disturbance of consciousness.

The hemiplegia following thrombosis or embolism has practically the characteristics, both primary and secondary, described under hæmorrhage.

The following may be the effects of blocking the different vessels:

(*a*) *Vertebral.*—The left branch is more frequently plugged. The effects are involvement of the nuclei in the medulla and symptoms of acute bulbar paralysis. It rarely occurs alone; more commonly with

(*b*) Blocking of the *basilar artery.* When this is entirely occluded

there may be bilateral paralysis from involvement of both motor paths. Bulbar symptoms may be present; rigidity or spasm may occur. The temperature may rise rapidly. The symptoms, in fact, are those of apoplexy of the pons.

(*c*) The *posterior cerebral* supplies the occipital lobe on its inner face and the greater part of the temporo-sphenoidal lobe. Localized areas of softening may exist without symptoms. Blocking of the branch passing to the cuneus may be followed by hemianopia. Hemianæsthesia may result from involvement of the posterior part of the internal capsule.

(*d*) *Internal Carotid.*—The symptoms are variable. As is well known, the vessel is in a majority of cases ligated without risk. In other instances transient hemiplegia follows; in others again the hemiplegia is permanent. These variations depend on the anastomoses in the circle of Willis. If these are large and free, no paralysis follows, but in cases in which the posterior communicating and the anterior communicating vessels are small or absent, the paralysis may persist. In No. 7 of my Elwyn series of cases of infantile hemiplegia, the woman, aged twenty-four, when six years old, had the right carotid ligated for abscess following scarlet fever, with the result of permanent hemiplegia. Blocking of the internal carotid within the skull by thrombosis or embolism is followed by hemiplegia, coma, and usually death. The clot is rarely confined to the carotid itself, but spreads into its branches and may involve the ophthalmic artery.

(*e*) *Middle Cerebral.*—This is the vessel most commonly involved, and, as already mentioned, if plugged before the central arteries are given off, permanent hemiplegia usually follows from softening of the internal capsule. Blocking of the branches beyond this point may be followed by hemiplegia, which is more likely to be transient, involves chiefly the arm and face, and if on the left side is associated with aphasia. The individual branches passing to the third frontal, ascending parietal, to the supramarginal and angular gyri, or to the temporal gyri may be plugged.

(*f*) *Anterior Cerebral.*—No symptoms may follow, and even when the branches which supply the paracental lobule and the top of the ascending convolutions are plugged the branches from the middle cerebral are usually able to effect a collateral circulation in these parts. Hebetude and dulness of intellect may occur with obstruction of the vessel.

There is unquestionably greater freedom of communication in the cortical branches of the different arteries than is usually admitted, although it is not possible, for example, to inject the posterior cerebral through the middle cerebral, or the middle cerebral from the anterior; but the absence of softening in some instances in which smaller branches are blocked shows how completely may be the compensation. The dilatation of the collateral branches may take place very rapidly; thus a patient with chronic nephritis died about twenty-four hours after the hemiplegic attack. There were recent vegetations on the mitral and an embolus

in the right middle cerebral artery just beyond the first two branches (temporal). The central portion of the hemisphere was swollen and œdematous. The right anterior cerebral was greatly dilated, and by measurement its diameter was found to be nearly three times that of the left.

Treatment of Cerebral Hæmorrhage.—The patient should be placed with the head high, and measures immediately taken to reduce the arterial pressure. Of these the most rapid and satisfactory is venesection, which should be practiced whenever the arterial tension is much increased. With a small pulse of low tension and signs of cardiac weakness it is contra-indicated. The chief difficulty is in determining whether the apoplexy is really due to hæmorrhage, or to thrombosis or embolism, since in the latter group of cases bleeding probably does harm. As a rule, however, in middle-aged men with arterio-sclerosis, an accentuated aortic second sound, and hypertrophy of the left ventricle, bleeding is indicated. Horsley and Spencer have recently, on experimental grounds, recommended the practice, formerly employed empirically, of compression of the carotid, particularly in the ingravescent form; or even, in suitable cases, passing a ligature round the vessel. An ice-bag may be placed on the head and hot bottles to the feet. The bowels should be freely opened, either by calomel, or croton oil placed on the tongue. Counter-irritation to the neck or to the feet is not necessary. When dyspnœa, stertor, and signs of mechanical obstruction are present, the patient should be turned on the side, as recommended by Bowles. This procedure also lessens the liability to congestion of the lungs.

Special care should be taken to avoid bed-sores; and if bottles are used to the feet, they should not be too hot, since blisters may be readily caused by much lower temperature than in health. In the fever of reaction, aconite may be indicated, but should be cautiously used. Stimulants are not necessary, unless the pulse becomes feeble and signs of collapse supervene.

The treatment of *softening* from thrombosis or embolism is very unsatisfactory. Venesection is not indicated, as it lowers the tension and rather promotes clotting. If, as is often the case, the heart's action is feeble and irregular, stimulants and small doses of digitalis may be given with, if necessary, ether or ammonia. The bowels should be kept open, but it is not well to purge actively, as in hæmorrhage.

In the thrombosis which follows syphilitic disease of the arteries, and which is met with most frequently in men between twenty and forty (in whom the hemiplegia often sets in without loss of consciousness), the iodide of potassium should be freely used, giving from twenty to thirty grains three times a day, or, if necessary, larger doses. If the syphilis has been recent, mercurials are also indicated. Practically these are the only cases of hemiplegia in which we see satisfactory results from treatment.

Operative treatment has been suggested, and when the diagnosis of

subdural hæmorrhage can be made it is justifiable. An attempt to reach a central hæmorrhage in the neighborhood of the internal capsule would only increase the damage to the brain-substance. Very little can be done for the hemiplegia which remains. The damage is too often irreparable and permanent, and it is very improbable that iodide of potassium, or any other remedy, hastens in the slightest degree Nature's dealing with the blood-clot.

The paralyzed limbs may be gently rubbed once or twice a day, and this should be systematically carried out, in order to maintain the nutrition of the muscles and to prevent, if possible, contractures. After the lapse of a fortnight the muscles may be stimulated by the faradic current; but when contractures develop, electricity is useless, and the passive movements and frictions are alone indicated.

In a case of complete hemiplegia, the friends should at the outset be frankly told that the chances of full recovery are slight. Power is usually restored in the leg sufficient to enable the patient to get about, but in the majority of instances the finer movements of the hand are permanently lost. The general health should be looked after, the bowels regulated, and the secretions of the skin and kidneys kept active. In permanent hemiplegia in persons above the middle period of life, more or less mental weakness is apt to follow the attack, and the patient may become irritable and emotional.

And, lastly, when hemiplegia has persisted for more than three months and contractures have developed, it is the duty of the physician to explain to the patient, or to his friends, that the condition is past relief, that medicines and electricity will do no good, and that there is no possible hope of cure.

Aneurism of the Cerebral Arteries.

Miliary aneurisms are not included, but reference is made only to aneurism of the larger branches. The condition is not uncommon. There were twelve instances in my first eight hundred autopsies in Montreal.* This is a considerably larger proportion than in Newton Pitt's collection from Guy's Hospital, nineteen times in nine thousand inspections.

Etiology.—Males are more frequently affected than females. Of my twelve cases seven were males. The disease is most common at the middle period of life. One of my cases was a lad of six. Pitt describes one at the same age. The chief causes are (*a*) endarteritis, either simple or syphilitic, which leads to weakness of the wall and dilatation; and (*b*) embolism. As pointed out by Church, these aneurisms are often found with endocarditis. Pitt, in his recent study of the subject, concludes that it is exceptional to find cerebral aneurism unassociated with fungating

* Canada Medical and Surgical Journal, vol. xiv.

endocarditis. The embolus disappears, and dilatation follows the secondary inflammatory changes in the coats of the vessel.

Morbid Anatomy.—The middle cerebral branches are most frequently involved. In my twelve cases the distribution on the arteries was as follows: Internal carotid, 1; middle cerebral, 5; basilar, 3; anterior communicating, 3. With the exception of one case they were saccular and communicated with the lumen of the vessel by an orifice smaller than the circumference of the sac. In the 154 cases which make up the statistics of Lebert, Durand, and Bartholow the middle cerebral was involved in 44, the basilar in 41, internal carotid in 23, anterior cerebral in 14, posterior communicating in 8, anterior communicating in 8, vertebral in 7, posterior cerebral in 6, inferior cerebellar in 3 (Gowers). The size of the aneurism varies from that of a pea to that of a walnut. The hæmorrhage may be entirely meningeal with very slight laceration of the brain substance, but the bleeding may be, as Coats has shown, entirely within the substance.

Symptoms.—The aneurism may attain considerable size and cause no symptoms. In a majority of the cases the first intimation is the rupture and the fatal apoplexy. Distinct symptoms are most frequently caused by aneurism of the internal carotid, which may compress the optic nerve or the commissure, causing neuritis or paralysis of the third nerve. A murmur may be audible on auscultation of the skull. Aneurism in this situation may give rise to irritative and pressure symptoms at the base of the brain or to hemianopsia. In the remarkable case reported by Weir Mitchell and Dercum an aneurism compressed the chiasma and produced bilateral temporal hemianopsia.

Aneurism of the vertebral or of the basilar may involve the nerves from the fifth to the twelfth. A large sac at the termination of the basilar may compress the third nerves or the crura.

The diagnosis is, as a rule, impossible. The larger sacs produce the symptoms of tumor, and their rupture is usually fatal.

Endarteritis.

In no group of vessels do we more frequently see chronic degenerative changes than in those of the circle of Willis. The condition occurs as:

(*a*) *Arterio-sclerosis*, producing localized or diffused thickening of the intima with the formation of atheromatous patches or areas of calcification. In the later stages, as seen in elderly people, the arteries of the circle of Willis may be dilated, stiff, or almost universally calcified.

(*b*) *Syphilitic Endarteritis.*—As already mentioned under the section of syphilis, gummatous endarteritis is specially prone to attack the cerebral vessels. It has in itself no specific characters—that is to say, it is impossible in given sections to pick out an endarteritis syphilitica from

an ordinary endarteritis obliterans. On the other hand, as already stated, the nodular periarteritis is never seen except in syphilis.

Thrombosis of the Cerebral Sinuses and Veins.

The condition may be primary or secondary.

Primary thrombosis of the sinuses and veins is rare. It occurs (*a*) in children, particularly during the first six months of life, usually in connection with diarrhœa. It has, in my experience, been a rare condition. I have never seen an example of spontaneous thrombosis of the sinuses in a child, and only two instances, both in connection with meningitis, in which the cortical veins contained clots. Gowers believes that it is of frequent occurrence, and that thrombosis of the veins is not an uncommon cause of infantile hemiplegia.

(*b*) In connection with chlorosis and anæmia. Brayton Ball has recently called attention to this interesting association, and has reported one case and collected ten or eleven others from the literature. All were in girls with anæmia or chlorosis.

(*c*) In the terminal stages of cancer, phthisis, and other chronic diseases thrombosis may gradually occur in the sinuses and cortical veins. To the coagulum developing in these conditions the term marantic thrombus is applied.

Secondary Thrombosis is much more frequent and follows extension of inflammation from contiguous parts to the sinus wall. The common causes are disease of the internal ear, fracture, compression of the sinuses by tumor, or suppurative disease outside the skull, particularly erysipelas. In these cases the lateral sinus is most frequently involved. Of 57 fatal cases in which ear-disease caused death with cerebral lesions, there were 22 in which thrombosis existed in the lateral sinuses (Pitt). The thrombus may be small, or may fill the entire sinus and extend into the internal jugular vein. In more than one half of these instances the thrombus was suppurating. The disease spreads directly from the necrosis on the posterior wall of the tympanum. It is not so common in disease of the mastoid cells.

Symptoms.—Primary thrombosis of the longitudinal sinus may occur without exciting symptoms and is found accidentally at the post-mortem. There may be mental dulness with headache. Convulsions and vomiting may occur. In other instances there is nothing distinctive. In a patient who died under my care, at the Philadelphia Hospital, of phthisis, there was a gradual torpor, deepening to coma, without convulsions, localizing symptoms, or optic neuritis. The condition was thought to be due to a terminal meningitis. In the chlorosis cases the head symptoms have, as a rule, been marked. Ball's patient was dull and stupid, had vomiting, dilatation of the pupils, and double choked disks. Slight paresis of the left side occurred. An interesting feature in her case was

the development of swelling of the left leg. In the cases reported by Andrew, Church, Tuckwell, Isambard Owen, and Wilks the patients had headache, vomiting, and delirium. Paralysis was not present. In Douglas Powell's case, with similar symptoms, there was loss of power on the left side. Bristowe reports a case of great interest in an anæmic girl of nineteen, who had convulsions, drowsiness, and vomiting. Tenderness and swelling developed in the position of the right internal jugular vein, and a few days later on the opposite side. The diagnosis was rendered definite by the occurrence of phlebitis in the veins of the right leg. The patient recovered.

The onset of such symptoms as have been mentioned in an anæmic or chlorotic girl should lead to the suspicion of cerebral thrombosis. In infants the diagnosis can rarely be made. Involvement of the cavernous sinus may cause œdema about the eyelids or prominence of the eyes.

In the *secondary thrombi* the symptoms are commonly those of septicæmia. For instance, in over seventy per cent of Pitt's cases the mode of death was by pulmonary pyæmia. This author draws the following important conclusions: (1) The disease spreads oftener from the posterior wall of the middle ear than from the mastoid cells. (2) The otorrhœa is generally of some standing, but not always. (3) The onset is sudden, the chief symptoms being pyrexia, rigors, pains in the occipital region and in the neck, associated with a septicæmic condition. (4) Well-marked optic neuritis may be present. (5) The appearance of acute local pulmonary mischief or of distant suppuration is almost conclusive of thrombosis. (6) The average duration is about three weeks, and death is generally from pulmonary pyæmia. The chief points in the diagnosis may be gathered from these statements.

Pitt records an interesting case of recovery in a boy of ten, who had otorrhœa for years and was admitted with fever, earache, tenderness, and œdema. A week later he had a rigor, and optic neuritis developed on the right side. The mastoid was explored unsuccessfully. The fever and chills persisting, two days later the lateral sinus was explored. A mass of foul clot was removed and the jugular vein was tied, after which the boy made a satisfactory recovery.

III. AFFECTIONS OF THE SUBSTANCE.

I. Topical Diagnosis.

A majority of the lesions of the nervous system which permit of a local diagnosis have as an important part of their symptomatology disturbance of muscular action, and as our knowledge of the mechanism governing the movements of muscles is comparatively exact, we shall take this system as a basis for local diagnosis.

The motor system is made up of two segments, each consisting of groups of nerve-cells, and their prolongations into nerve-fibres. The *upper segment* comprises the motor cortex and the pryamidal fibres; and the *lower segment* the motor cells in the medulla and cord and the nerve-fibres arising from them, forming the peripheral nerves distributed to the muscles, which may themselves be considered as part of this segment.

The nerve-cells are so arranged that when thrown into action, by whatever cause, a definite movement is the result, and the same combination of nerve-cells always causes the same movement, or, in other words, every movement of the body is represented in the nervous centres by combinations of the nerve-cells, or, as we say, is localized.

Movements are localized both in the cells of the lower segment and in those of the upper, and we have consequently spinal localization and cerebral localization.

Spinal Localization.—In the lower motor segment the muscles are represented in their simplest movements, and different sections of the cord have been found to represent the movements of different muscles. Our knowledge of this localization is by no means complete, but enough has been learned to aid us materially in determining the site of a spinal lesion.

The cells of the lower segment are found in the motor nuclei of the medulla, and in the anterior gray horns of the spinal cord. They are connected with the muscles by the axis cylinder processes, the anterior nerve-roots (roots of motor cranial nerves), the peripheral nerves, and the end organs by which they are brought into intimate relation with the protoplasm of the muscle fibre itself.

The following table prepared by Starr gives in detail our knowledge on this subject:

Localization of the Functions of the Segments of the Spinal Cord.

Segment.	Muscles.	Reflex.	Sensation.
II and III C.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypchondrium (f). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.

Segment.	Muscles.	Reflex.	Sensation.
IV C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra and infra spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis(clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI C.	Biceps. Brachialis anticus. Pectoralis(clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latisimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I D.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.
II to XII D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th dorsal. Tickling mammary regions causes retraction of epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen in bands running around and downward, corresponding to spinal nerves. Upper gluteal region.
I L.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 1st to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.

Segment.	Muscles.	Reflex.	Sensation.
II L.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patella tendon. Stroking tendon causes extension of leg.	Outer side of thigh.
III L.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV L.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V L.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I to II S.	Flexors of ankle. Long flexor of toes. Peronæi. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
III to V S.	Perineal muscles.	Foot reflex. Achilles tendon. Overextension of foot causes rapid flexion; ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perinæum. Genitals.

Cerebral Motor Localization.—In the motor cortex the muscles are again represented, or, as Hughlings Jackson says, re-represented in their finer movements.

Motor Centres.—The experiments of Hitzig and Fritsch and of Ferrier, together with the previous clinical studies of Hughlings Jackson, laid the foundation of our present knowledge of cerebral localization.

The area for representation of the movements in the cerebral cortex is in the Rolandic region and comprises the ascending parietal and ascending frontal convolutions, the hinder part of the three frontal convolutions, and the parietal lobule, a continuation backward of the ascending parietal convolution (Fig. 2, motor region). This entire region is excitable, and stimulation by weak electrical currents produces muscular movements in the opposite half of the body. The centres presiding over the different groups of muscles may be thus classified:

(*a*) Centres for the trunk. These have been shown by Schäfer to be situated in the marginal gyrus, just within the longitudinal fissure, the region sometimes spoken of as the paracental lobule.

(*b*) Centres for the lower limbs. These are situated at the upper part of the Rolandic region, close to the longitudinal fissure. As indicated in the diagram, the representation of movements of the different portions of the lower limb in this region is as follows (Fig. 2): Most anterior, the hip; next in order, the knee and ankle; then the big toe, the centre for

the movement of which surrounds the upper end of the fissure of Rolando. Still further back are the centres for movement of the small toes.

(*c*) Centres for the upper limbs. This area corresponds to about the

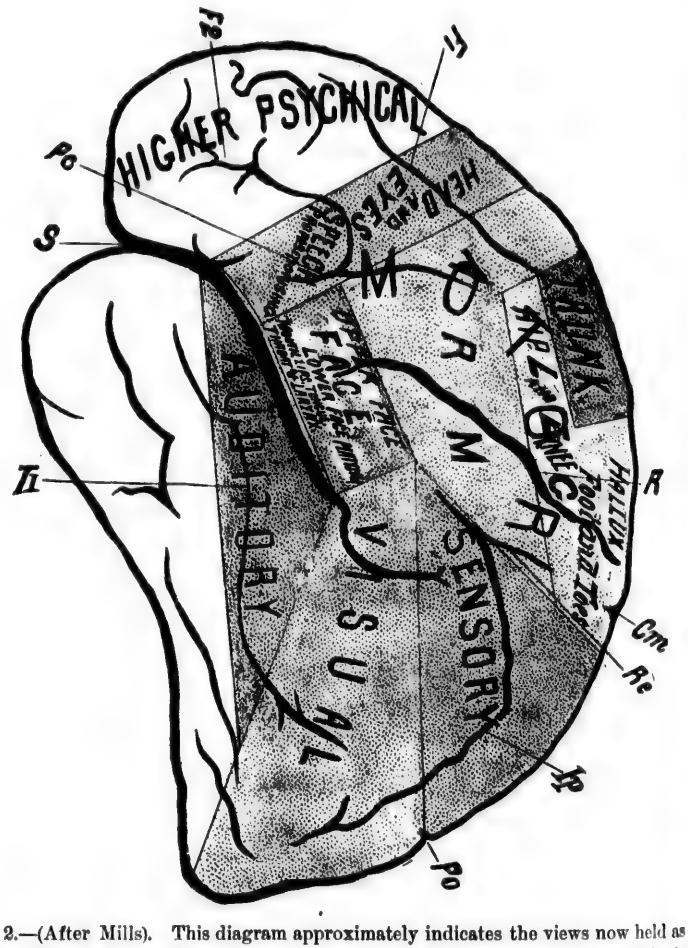


FIG. 2.—(After Mills). This diagram approximately indicates the views now held as a result of experiment and their confirmation or modification by clinico-pathological observation. It represents the division of the lateral surface of the cerebrum into higher psychical, motor, sensory, visual, and auditory areas; also the subdivision of the motor area into subareas for speech, the head and eyes, the face, arm, leg, and trunk. Only certain main points have been indicated by lettering, so as not to confuse: *S*, fissure of Sylvius; *R*, fissure of Rolando, or central fissure; *Pc*, precentral fissure; *Rc*, retrocentral fissure; *F1*, *F2*, superior and inferior frontal fissure; *Ip*, interparietal fissure; *Po*, parieto-occipital fissure; *T1*, first temporal fissure.

middle two fourths of the motor area. The careful studies of Horsley and Beevor have shown that from above downward the different segments of the limbs are represented as follows: Shoulder, elbow, wrist, fingers, the index-finger, and, lowest of all, the thumb.

(*d*) The centres for the face, tongue, pharynx, and larynx are situated in the lowest portion of the Rolandic area. The centres for the movement of the tongue and vocal cords are in the lower and anterior portion of the ascending convolution, and on the left side in man this region and the posterior part of the third left frontal convolution constitute the speech centre (Fig. 2), destruction of which is followed by one form of aphasia. In front of the precentral sulcus are centres for the representation of movements for turning the head and eyes to the opposite side.

The determination of these areas was worked out in animals and has now been thoroughly established in man, both by clinical observation and by the application of the electrodes in different situations during operations for the removal of growths in the brain or of the motor centres in epilepsy. The different regions must not be regarded as sharply separated from, but as blending with each other.

With these centres for voluntary movements are associated those which preside over the muscular sense, which is a compound of sensory impressions, of pressure, tension, and touch derived from the muscles as they are in motion. There is still dispute with reference to the localization of this sense, but the general opinion is that lesions of the motor area itself cause slight loss both of muscular and tactile sense. Others place the centres for general sensation in the situation marked in Fig. 2.

The fibres uniting the cortical motor centres and the spinal centres have a long course, in which they probably have no connection with any other nerve-cells. They arise from the various centres, enter the white matter of the hemisphere (the corona radiata), and gradually converge to what is called the internal capsule, which lies between the lenticular nucleus and the thalamus and the caudate nucleus (Fig. 3). The position of the fibres in the internal capsule has been accurately worked out by several observers. The fibres from the centres of the face, tongue, eyes, and head occupy the most anterior position, just at the knee, as it is called, of the internal capsule, while the fibres from the upper extremities are just behind these, and those from the lower extremities occupy the position in the middle third of the posterior part. Leaving the internal capsule, the fibres forming the motor path pass from the brain into the crus, in which they occupy a lower and medial position. Passing through the pons, covered by the superficial layers of transverse fibres, they enter the medulla, of which they form the anterior or pyramidal tract. At the lower part of the medulla a large proportion of the fibres decussate and pass into the opposite side of the spinal cord, forming the crossed pyramidal tract of the lateral column, while a smaller number of the fibres descend in the anterior column of the same side, forming the direct pyramidal tract, or

Türck's column. The pyramidal tracts diminish in size from above downward. The fibres enter the gray matter between the anterior and

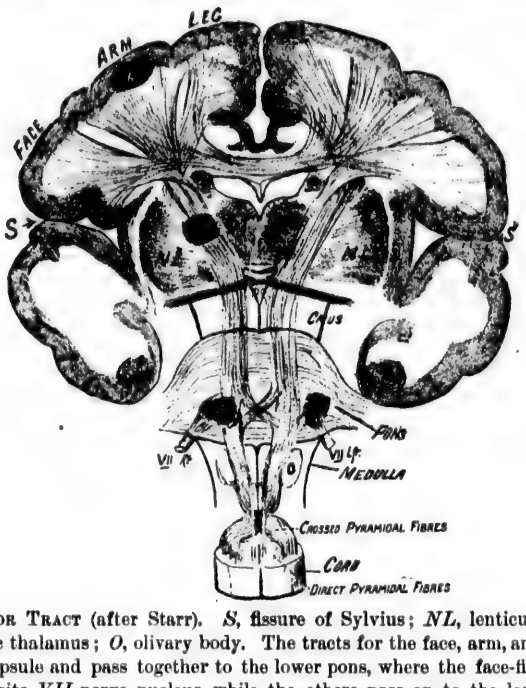


FIG. 3.—MOTOR TRACT (after Starr). *S*, fissure of Sylvius; *NL*, lenticular nucleus; *OT*, optic thalamus; *O*, olivary body. The tracts for the face, arm, and leg gather in the capsule and pass together to the lower pons, where the face-fibres cross to the opposite *VII* nerve nucleus, while the others pass on to the lower medulla, where they partially decussate to enter the lateral columns of the cord; the non-decussating fibres pass to the anterior median columns. The effect of a lesion situated at three points in the tract is shown on the left side of the figure at *X*, *Y*, *Z*. At *Z* the lesion would involve the left facial nerve and the left pyramidal tract above the decussation, producing facial paralysis on the left side and paralysis of the arm and leg on the opposite side—crossed paralysis.

posterior cornua, pass forward, divide and subdivide, and finally join the plexus of the protoplasmic processes, and are in this way connected with the large nerve-cells of the anterior horns.

Lesions of the Motor System.—Each of the segments of the motor tract is to be considered as a nutritional unit, depending for its vitality upon the integrity of its ganglion cells. If certain cells in the cortex are destroyed, the fibres arising from them will degenerate throughout their length—that is, to the beginning of the lower motor segment. So also if the motor cells in the medulla or cord are injured, their nerve-fibres will degenerate, and the muscles to which they are distributed will also be involved in the process. The same thing occurs if the nerve-fibres become detached from their ganglion cells. This process is called secondary

degeneration or Wallerian degeneration, after the physician who first described it. Fig. 4 illustrates this process in the cortico-spinal motor segment.

The lesions may be grouped, as Hughlings Jackson suggested, into negative and positive, or, as they are now more usually termed, destructive and irritative.

Negative or destructive lesions anywhere in the motor path have as a result the abolishment of the functions of these parts—i. e., *paralysis*.

Positive or irritative lesions cause a perversion of the function—i. e., *abnormal muscular contractions*.

Although these two symptoms (paralysis and abnormal contractions) occur whenever the motor path is diseased, each of the segments imparts to them peculiar characteristics which enable us in a great majority of cases to determine the site of a lesion.

These characteristics depend upon, first, the special symptoms referable to the secondary degenerations in the two segments; second, upon their anatomical relation.

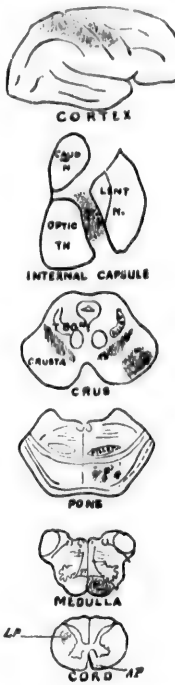


FIG. 4. — (After Gowers.) Diagram showing course and degeneration of pyramidal tract in right hemisphere, crus, pons medulla, and cord.

(*a*) **Lesions of the Lower or Spino-muscular Segment.** *Destructive Lesions.*—The destructive lesions cause here, as everywhere in the motor path, paralysis. We have seen above that when the nerve-fibres are cut off from their ganglion cells in the anterior horns, they not only degenerate themselves, but that the muscles to which they are distributed degenerate. This process is made evident by a change in the electrical reaction of the nerve and muscle—the reaction of degeneration—and the muscle becomes evidently atrophied. The myotatic irritability or muscle reflex, which depends upon the integrity of the lower motor segment, is lost in destructive lesions. This gives to the paralysis certain characteristics, namely, atrophy of the muscles, loss of its reflex excitability, and alteration of the electrical reactions of the nerve and muscle.

The anatomical relations of the lower motor segment also give certain peculiarities, which help to distinguish its lesions from those of the upper segment, on the one hand, and of the different parts of the lower segment on the other.

In general the different units which make up the lower segment are more or less widely separated from each other. An extreme example of

this is the distance between the nucleus of the third nerve and the collection of motor cells in the lower part of the lumbar enlargement. For this reason lesions of this segment are more apt to cause paralysis of individual muscles or muscle groups, as distinguished from the more wide-spread paralysis due to lesions of the upper segment.

Reference to Starr's table will show that the muscles are represented in the spinal cord without relation to the nerves which supply them—that is to say, muscles that are supplied by a certain nerve may not be represented close together in the anterior horns; for instance, in the fourth cervical segment, movements of the diaphragm, deltoid, biceps, supinator longus, rhomboid, supraspinatus, and infraspinatus are represented. It follows from this that the distribution of a paralysis due to disease of the lower motor segment may enable us to distinguish the position of the lesion within the segment itself. We are often helped in this by the sensory symptoms, which may accompany the paralysis. Thus, if we have a paralysis with the characteristics of a lesion of the lower motor segment, and if the paralyzed muscles are all supplied by one nerve, and we discover anæsthesia in the skin of the arm supplied by that nerve, it is evident that the lesions must be in the nerve itself. On the other hand, if the muscles paralyzed are not supplied by a single nerve, but are represented close together in the spinal cord, and the anæsthesia corresponds to that section of the cord (see table), it is equally clear that the lesion must be of the cord itself or of its nerve-roots.

Irritative Lesions of the Lower Motor Segment.—We know of no lesion of this segment which has as its result abnormal muscular contraction unless the slow atrophy of the ganglion cells occurring in progressive muscular atrophy be considered as the cause of the fibrillary contraction so common in this affection.

(Certain tonic muscular contractions occurring in poisoning by strychnine and in tetanus are thought to be due to the perverted action of the lower motor centres, and Hughlings Jackson believes that certain convulsive paroxysms—"lowest level fits"—are due to discharging lesions of these centres, and claims laryngismus stridulus in this category.)

(*b*) **Lesions of the Upper, Cerebro-spinal Motor Segment.**—*Destructive lesions* cause, as in the lower motor segment, paralysis, and here again the secondary degeneration which follows the lesion gives to the paralysis its distinctive characteristics. In this case the paralysis is accompanied by a spastic condition, shown in an exaggeration of muscle reflex and an increase in the tension of the muscle. It is not accurately known how the degeneration of the pyramidal fibres causes this excess of the muscle reflex. The usual explanation is that under normal circumstances the upper motor centres are constantly exerting a restraining influence upon the activity of the lower centres, and that when the influence ceases to act, on account of disease of the pyramidal fibres, the latter take on increased activity, which is made manifest by an exaggeration of the muscle reflex.

It was stated above that each segment of the motor path is to be considered as a nutritional unit and that the secondary degeneration in the upper segment stops at the beginning of the lower. So the muscles paralyzed by lesions in the upper segment do not undergo degenerative atrophy, nor do they present the reaction of degeneration.

The upper motor segment is much more compact than the lower, and for this reason a paralysis resulting from a lesion in it is apt to involve many muscles. This is especially true in regard to the pyramidal fibres, which run in a compact bundle, a lesion of which usually involves all of the fibres and causes a paralysis of all of the muscles of one side of the body—i. e., *hemiplegia*.

The motor centres of the cortex are more or less separated from each other, and a sharply localized lesion in this region causes a more limited paralysis, and cerebral monoplegias are the result; but even in this case the paralysis is diffuse, affecting the whole limb or a segment of the limb, and not individual muscles or groups of muscles.

To sum up, the paralyses due to lesions of the cerebro-spinal motor segment are diffuse, wide-spread, often hemiplegic; the paralyzed muscles are spastic (the tendon reflexes exaggerated), they do not undergo degenerative atrophy, and they do not present the degenerative reaction to electrical stimulation.

Irritative Lesions of the Upper Motor Segment.—Our knowledge of such lesions is confined for the most part to those acting on the cortical motor centres, and we know a number of processes which have as their result abnormal muscular contractions. These have as their type the localized convulsive seizures classed under Jacksonian or cortical epilepsy, which are characterized by the convulsion beginning in a single muscle or group of muscles and involving other muscles in a definite order, depending upon the position of their representation in the cortex; for instance, such a convulsion beginning in the muscles of the face next involves those of the arm and hand, and then the leg. The convulsion is usually accompanied by sensory phenomena and followed by a weakness of the muscles involved.

A majority of lesions of the motor cortex are both destructive and irritative—i. e., they may destroy the nerve-cells of a certain centre, and either by their growth or presence may throw into abnormal activity those of the surrounding centres.

So far the motor system has been considered by itself, and we have endeavored to show how attention to the paralysis alone may help us to determine the seat of a lesion. It runs, however, in close connection with other systems of the nervous centre, which are often involved with it in morbid processes, giving rise to symptoms which aid us very much in making a local diagnosis.

Sensory Centres and Paths.—The association of the motor path with that for the conduction of sensory impressions is very intimate, but unfortunately our knowledge of the exact position of the sensory tracts is by

no means so precise. Some important facts are, however, known. Sensory fibres from different areas of the skin run in close connection with fibres of the lower motor segment in the mixed nerves. They separate from them and enter the spinal cord by the posterior roots. The regions which the different posterior roots supply is given in Starr's table. After entering the spinal cord the sensory fibres cross the middle line at once and pass up to the brain in the opposite half of the cord. Here they are again in close contact with the motor path, but with that of the other side of the body—i. e., the right half of the spinal cord contains the sensory fibres of the left side of the body and motor fibres of the right. The fibres which conduct the impressions for the muscular sense seem to be an exception and do not decussate in the cord. The exact position of the sensory paths in the cord is still somewhat uncertain, nor are we sure of their course in the medulla, pons, and peduncle. All the sensory fibres of the opposite side of the body are collected in the posterior third of the posterior limb of the internal capsule, just behind the motor fibres of the upper segment.

Much doubt and discussion still exist as to the areas for the representation of sensory impressions. Horsley has suggested that the muscular and tactile senses are localized in the motor cortex, and that two of the three chief layers of cells in this region subserve their functions. Dana's study shows that many lesions of the motor area, particularly in the hinder part, are associated with anæsthesia. On the other hand, Ferrier regards the hippocampal convolution, and Schäfer the gyrus formicatus, as the centres for sensory impressions.

The centres for sight, hearing, smell, and taste have been referred to under the nerves ministering to these senses, and we shall consider the speech centres in the next section.

In the centrum ovale the fibres of the motor path are more or less closely associated with other systems of fibres; those connecting the cortex with nervous structures lying below it, projection fibres; the fibres which join the two hemispheres, commissural fibres; and those which join different parts of the same hemisphere, association fibres. Our knowledge of the function of these fibres leaves much to be desired.*

The following is a brief summary of the effects of lesions from the cortex to the spinal cord:

1. **The Cerebral Cortex.**—(*a*) Destructive lesions cause *spastic paralysis* in the muscles of the opposite side of the body. The extent of the paralysis depends upon that of the lesion. It is apt to be limited to the muscles of an extremity, giving rise to the cerebral monoplegias (Fig. 3, *X*). A lesion may involve two centres lying close together, thus producing paralysis of the face and arm, or of the arm and leg, but not of

* The student will find in Starr's work, Familiar Forms of Nervous Disease, an admirable presentation of this subject.

the face and leg without involvement of the arm. Very rarely the whole motor cortex is involved, causing paralysis of one side—cortical hemiplegia.

Combined with the muscular weakness there is usually some disturbance of sensation, particularly tactile impressions and those of the muscular sense.

(*b*) Irritative lesions cause localized spasms as described above. These convulsions are usually preceded and accompanied by sensory impressions. Tingling or pain, or a sense of motion in the part, is often the *signal symptom* (Seguin), and is of great importance in determining the seat of the lesion.

Lesions are often both destructive and irritative, and we have combinations of the symptoms produced by each. For instance, certain muscles may be paralyzed, and those represented near them in the cortex may be the seat of localized convulsions, or the paralyzed limb itself may be at times subject to convulsive spasms, or muscles which have been convulsed may become paralyzed. In this manner it is often possible to trace the progress of a lesion involving the motor cortex.

We have seen in a previous section that lesions involving the centres for the special senses may give rise to focal symptoms, and shall simply refer to them here. The symptoms caused by lesions of the speech centre will be described under aphasia, and it is only necessary to note the near situation of the motor speech area (Broca's centre) in the left third frontal convolution to the centres of the face and arm on that side, and to state that motor aphasia is often associated with monoplegia of the right side of the face and the right arm. Accompanying the paralysis following a Jacksonian fit of the right face or arm there is often a transient motor aphasia.

(2) **Centrum Ovale.**—Lesions in this part of the motor path cause paralysis, which has the distribution of a cortical palsy when the lesion is near the cortex, and of that due to a lesion of the internal capsule when it is near that region. They may be associated with symptoms due to the interruption of the other system of fibres running in the centrum ovale, and there may be sensory disturbances—hemianæsthesia and hemianopia—and if the lesion is in the left hemisphere one of the different forms of aphasia may accompany the paralysis.

(3) **Internal Capsule.**—Here all the fibres of the upper motor segment are gathered together in a compact bundle, and a lesion in this region is apt to cause complete hemiplegia of the opposite side, and if the lesion involves the hinder third of the posterior limb there is also hemianæsthesia, including even the special senses(Fig. 3, *Y*).

(4) **Crus Cerebri.**—Here, again, all the motor fibres and all the sensory fibres of the opposite side are collected in a small space, and a lesion may produce hemiplegia combined with sensory disturbances. On account of its anatomical relation the third cranial nerve is often involved

in lesions of the crus, causing paralysis of the muscles of the eye on the same side as the lesion combined with a hemiplegia of the opposite side—i. e., a crossed paralysis.

(5) **Pons.**—In the pons, medulla, and cord the upper and lower motor segments are both represented, the first by the pyramidal fibres, the latter by the motor nuclei and the nerve-fibres arising from them. Lesions here often affect both motor segments, and produce combinations of paralyses having the characteristics of each. Thus a lesion in the lower part of the pons may involve the pyramidal tract and cause a spastic paralysis of the opposite arm and leg, and also involve the nucleus or the fibres of the facial nerve, and so produce a paralysis of the same side of the face, accompanied by loss of the muscle reflex, atrophy, and the reaction of degeneration—crossed paralysis (Fig. 3, *Z*). The abducens and hypoglossus nerves may also be paralyzed in the same manner. In lesions of the pons the patient often has a tendency to fall toward the side on which the lesion is, due probably to implication of the middle peduncle of the cerebellum.

The symptoms produced by involvement of the different cranial nerves have been considered in detail in a previous section.

(6) **Spinal Cord.**—Unilateral lesions cause, first, a lower-segment paralysis, due to the disease of the centres at the site involved; second, a spastic paralysis of all the muscles on that side of the body below the lesion, due to interruption of the pyramidal fibres; and, third, disturbance of sensation in the opposite side of the body. (See under Brown-Séquard's paralysis.)

Transverse lesions of the cord cause paralysis with atrophy, etc., at the level of the lesion, spastic paralysis below it, combined with sensory disturbance and trouble with the bladder and rectum.

Affections of the peripheral nerves have already been considered.

II. Aphasia.

The speech mechanism consists of receptive, perceptive, and emissive centres in the cortex cerebri, disturbances of which cause *aphasia*, and centres in the medulla which preside over the muscles of articulation, disturbance of which produces *anarthria*, the condition of gradual loss of power of speech, such as occurs in bulbar paralysis.

The studies of Bastian, Küssmaul, Wernicke, Lichtheim, and others have widened enormously our knowledge of speech disorders. Language is gradually acquired by imitation. Thus, in teaching a child to say *bell*, the sound of the uttered word enters the afferent path (auditory nerve) and reaches the auditory perceptive centre, from which an impulse is sent to the emissive or motor centre presiding over the nuclei in the medulla, through which the muscles of articulation are set in action. The arc in Lichtheim's schema (Fig. 5) is a A, Mm. The child gradually ac-

quires in this way *word memories*, which are stored at the centre A, and *motor memories*—the memories of the co-ordinated muscular movements necessary to utter words—which are stored at the centre M. In a similar manner, when shown the bell, the child acquires *visual memories*, which are conveyed through the optic nerve to the visual perceptive centres, o O. So also the memories of the sound of the bell when struck. The memory picture of the shape of the bell, the memory of the appearance of the word bell as written, and the motor memories of the muscular movements required to write the word are distinct from each other; yet they are intimately connected, and form together what is termed the *word-image*. In addition to all this the child gradually acquires in his education ideas as to the use of the bell—intellectual concepts—the centre for which is represented at I in the diagram In volitional or intellectual speech, as in uttering the word *bell*, the path would be I, M m, and in writing the word, I, M, W, h. These various "memories" are as a rule stored or centred in the left hemisphere.

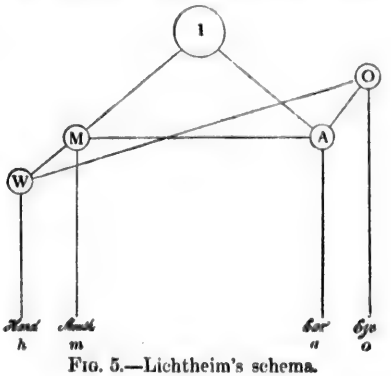


FIG. 5.—Lichtheim's schema.

The relations of written and spoken language are then with (*a*) sensory perceptive centres (hearing and sight and, in the blind, touch); (*b*) emissive or motor centres for speech and writing; and (*c*) psychical centres, through which we obtain an intellectual conception of what is said or written, and by which we express voluntarily our ideas in language.

There are two chief forms of aphasia—*sensory* and *motor*.

(1) **Sensory Aphasia; Apraxia; Word-blindness; Word-deafness.**—By apraxia is understood a condition in which there is loss or impairment of the power to recognize the nature and characteristics of objects. Persons so affected act "as if they no longer possessed such object memories, for they fail to recognize things formerly familiar. A fork, a cane, a pin, may be taken up and looked at by such a person, and yet held or used in a manner which clearly shows that it awakens no idea of its use. And this symptom, for which at first the term blindness of mind was used, is found to extend to other senses than that of sight. Thus the tick of a watch, the sound of a bell, a melody of music, may fail to arouse the idea which it formerly awakened, and the patient has then deafness of mind, or an odor or taste no longer calls up the notion of the thing smelled or tasted; and thus it is found that each or all of the sensory organs, when

called into play, may fail to arouse an intelligent perception of the object exciting them. For the general symptoms of inability to recognize the use or import of an object the term *apraxia* is now employed." (Starr.)

Apraxia may occur alone, but more commonly is associated with varieties of sensory and motor aphasia. The patient may be able to read, but the words arouse no intelligent impression in his mind. While blind to memory-pictures aroused through sight, the perceptions may be stimulated by touch; thus there are instances on record of apraxic patients unable to read by sight, who could on tracing the letters by touch name them correctly. Of the forms of apraxia, mind-blindness and mind-deafness are the most important.

The cases of *mind-blindness* collected by Starr indicate that the lesion exists in the left hemisphere in right-handed persons, and in the right hemisphere in left-handed persons. The disease usually involves the angular and supramarginal gyri or the tracts proceeding from them. Blindness of the "mind's eye" may at times be functional and transitory, and is associated with many forms of mental disturbance. In a remarkable case reported by Macewen, the patient, after an injury to the head, had suffered with headache and melancholia, but there was no paralysis. He was psychically blind and though he could see everything perfectly well and could read letters, objects conveyed no intelligent impression. A man before his eyes was recognized as some object, but not as a man until the sounds of the voice led to the recognition through the auditory centres. The skull was trephined over the angular gyrus and the inner table was found to be depressed and a portion had been driven into the brain in this region. The patient recovered. Mind-blindness is the equivalent of visual amnesia.

Word-blindness may occur alone or with motor aphasia. In uncomplicated cases the patient is no longer able to recall the appearances of words, and does not recognize them on a printed or written page. The patient may be able to pronounce the letters and can often write correctly, but he cannot read understandingly what he has written. It is rare, however, for the patient to be able to write with any degree of facility. There are instances in which the patient, unable to read, has yet been able to do mathematical problems and to recognize play cards. The lesion in cases of word-blindness is, in a majority of cases, in the angular and supramarginal gyri on the left side. It is commonly associated with hemianopia, and not infrequently with mind-blindness.

Mind-deafness is a condition in which sounds, though heard and perceived as such, awaken no intelligent conceptions. A person who knows nothing of French has mind-deafness so far as the French language is concerned, and though he recognizes the words as words when spoken, and can repeat them, they awaken no auditory memories. The musical

faculties may be lost in aphasics, who may become note-deaf and unable to appreciate melodies or to read music. This may occur without the existence of motor aphasia, and on the other hand there are cases on record in which with motor aphasia for ordinary speech the patient could sing and follow tunes correctly. Mind-deafness is also known as auditory amnesia. Word-deafness is a condition in which the patient no longer understands spoken language. The memory of the sound of the word is lost, and can neither be recalled nor recognized when heard. It is usually associated with other varieties of aphasia, though there are cases in which the patient has been able to read and write and speak. The lesion in word-deafness has been accurately defined in a number of cases to be in the posterior portion of the first and second temporal convolutions on the left side (Fig. 2).

Other manifestations of mind-blindness are met with; thus a young man with secondary syphilis had several convulsive seizures, after one of which he remained unconscious for some time. On awakening, the memory-pictures of faces and places were a blank, and he neither knew his parents nor brothers, nor the streets of the town in which he lived. He had no aphasia proper, and no paralysis.

(2) **Motor or ataxic aphasia** is a condition in which the memory of the efforts necessary to pronounce words is lost, owing to disturbance in the emissive centres. This is the variety long ago recognized by Broca, the lesion of which was localized by him in the third left frontal convolution. In pure cases the patient is able to read (not aloud) and understands perfectly what is said. He may not be able to utter a single word; more commonly he can say one or two words, such as "no," "yes," and he not infrequently is able to repeat words. When shown an object, though not able to name it, he may evidently recognize what it is. If told the name, he may be able to repeat it. A man knowing the French and German languages may lose the power of expressing his thoughts in them, while retaining his mother-tongue; or, if completely aphasic, may recover one before the other. As the third left frontal convolution is in close contact with the centres for the face and arm, these are not uncommonly involved, with the production of a partial or, in some instances, a complete right-sided hemiplegia. *Alexia*, or inability to read, occurs with motor aphasia and also with word-blindness.

As a rule, in motor aphasia there is also inability to write—*agraphia*. When there is right brachial monoplegia it is difficult to test the capability, but there are instances of motor aphasia without paralysis, in which the power of voluntary writing is lost. The condition varies very much; thus a patient may not be able to write voluntarily or from dictation, and yet may copy perfectly. It is still a question whether there is a special writing centre. It has been placed by some writers at the base of the second frontal convolution, but in a

recent study Déjérine concludes that it is not separate from the speech centre.

There is a form known as *mixed aphasia*, or *paraphasia*, in which the patient understands what is said, and speaks even long sentences correctly, but he constantly tends to misplace words, and does not express his ideas in the proper words. All grades of this may be met with, from a state in which only a word or two is misplaced to an extreme condition in which the patient talks jargon. In these cases the association tract is interrupted between the auditory perceptive and the emissive centres, hence it is sometimes known as Wernicke's aphasia of conduction. The lesion is usually in the insula and in the convolutions which unite the frontal and temporal lobes. Lichtheim's schema will assist the student in obtaining a rational idea of the varieties of aphasia:

1. In the condition of apraxia or mind-blindness the ideation centres, I, are involved, often with the auditory and visual perceptive centres, A and O.

2. A lesion at A, the centre for the auditory memories of words (first left temporal gyrus), is associated with word-deafness.

3. A lesion at O, the centre for visual memories (angular and supramarginal gyri), causes word-blindness.

4. Interruption of the tracts uniting A M and O M causes the conduction aphasia of Wernicke—*paraphasia.*

5. Destruction of the centre M (Broca's convolution) causes pure motor aphasia, in which the patient cannot express thoughts in speech.

A lesion at M usually destroys also the power of writing, but, as stated, it is believed by many that the centre for writing, W, is distinct from that of speech. In this case a lesion at M, which would destroy the power of voluntary speech, might leave open the connections between O W and A W, by which the patient could copy or write from dictation.

The problems of aphasia are in reality excessively complicated, and the student must not for a moment suppose that cases are as simple as diagrams indicate. A majority of them are very complex, but with patience the diagnosis of the different varieties can often be worked out.

The following tests should be applied in each case of aphasia: (1) The power of recognizing the nature, uses, and relations of objects—i. e., whether apraxia is present or not; (2) the power to recall the name of familiar objects seen, smelled, or tasted, or of a sound when heard, or of an object touched; (3) the power to understand spoken words; (4) the capability of understanding printed or written language; (5) the power of appreciating and understanding musical tunes; (6) the power of voluntary speech—in this it is to be noted particularly whether he mis-

places words or not; (7) the power of reading aloud and of understanding what he reads; (8) the power to write voluntarily and of reading what he has written; (9) the power to copy; (10) the power to write at dictation; and (11) the power of repeating words.

Prognosis and Treatment.—In young persons the outlook is good, and the power of speech is gradually restored apparently by the education of the centres on the opposite side of the brain. In adults the condition is less hopeful, particularly in the cases of complete motor aphasia with right hemiplegia. The patient may remain speechless, though capable of understanding everything, and attempts at re-education may be futile. Partial recovery may occur, and the patient may be able to talk, but misplaces words. In sensory aphasia the condition may be only transient, and the different forms rarely persist alone without impairment of the powers of expression.

The education of an aphasic person requires the greatest care and patience, particularly if, as so often happens, he is emotional and irritable. It is best to begin by the use of detached letters, and advance, not too rapidly, to words of only one syllable. Children often make rapid progress, but in adults failure is only two frequent, even after the most pains-taking efforts. In the cases of right hemiplegia with aphasia the patient may be taught to write with the left hand.

III. Inflammation of the Brain

(Suppurative Encephalitis; Abscess).

Etiology.—Suppuration of the brain substance is rarely if ever primary, but results, as a rule, from extension of inflammation from neighboring parts or infection from a distance through the blood. The question of idiopathic brain abscess need scarcely be considered, though occasionally instances occur in which it is extremely difficult to assign a cause. There are three important etiological factors:

(1) Trauma. Falls upon the head or blows, with or without abrasion of the skin. More commonly it follows fracture or punctured wounds. In this group meningitis is frequently associated with the abscess.

(2) Extension of the inflammation from the neighboring parts, more particularly in caries of the petrous portion of the temporal bone, less frequently necrosis of the other bones, or extension of disease from the orbit. In this group otitis is the most important factor. There may be extension through the bone and involvement of the lateral sinus as already mentioned; but in other instances no direct connection can be traced and the infection is probably carried through the lymph channels.

(3) In septic processes. Abscess of the brain is not often found in pyæmia. In ulcerative endocarditis multiple foci of suppuration are

common. Localized bone-disease, suppuration in the liver, but, above all, certain inflammations in the lungs (particularly gangrene, bronchiectasis, and fetid bronchitis), are liable to be followed by abscess. It is an occasional complication of empyema. Abscess of the brain may follow the specific fevers. Bristowe has called attention to its occurrence as a sequel of influenza. The largest number of cases occur between the twentieth and fortieth years, and the condition is more frequent in men than in women.

Morbid Anatomy.—The abscess may be solitary or multiple, diffuse or circumscribed. In the acute, rapidly fatal cases following injury the suppuration is not limited; but in long-standing cases the abscess is enclosed in a definite capsule, which may have a thickness of from two to five millimetres. The pus varies much in appearance, depending upon the age of the abscess. In early cases it may be mixed with reddish *débris* and softened brain matter, but in the solitary encapsulated abscess the pus is distinctive, having a greenish tint, an acid reaction, and a peculiar odor, sometimes like sulphuretted hydrogen. The brain substance surrounding the abscess is usually œdematous and infiltrated. The size varies from that of a walnut to that of a large orange. There are cases on record in which the cavity has occupied the greater portion of a hemisphere. Multiple abscesses are usually small. In four fifths of all cases the abscess is solitary. Suppuration occurs most frequently in the cerebrum, and the temporo-sphenoidal lobe is more often involved than other parts. The cerebellum is the next most common seat, particularly in connection with ear-disease.

Symptoms.—Following injury or operation the disease may run an *acute* course, with fever, headache, delirium, vomiting, and rigors. The symptoms are those of an acute meningo-encephalitis, and it may be very difficult to determine, unless there are localizing symptoms, whether there is really suppuration in the brain substance. In the cases following ear disease the symptoms may at first be those of meningeal irritation. There may be irritability, restlessness, severe headache, and aggravated earache. Other striking symptoms, particularly in the more prolonged cases, are drowsiness, slow cerebration, vomiting, and optic neuritis. In the chronic form of brain abscess which may follow injury, otorrhœa, or local lung trouble, there may be a latent period ranging from one or two weeks to several months, or even a year or more. In the "silent" regions, when the abscess becomes encapsulated there may be no symptoms whatever during the latent period. During all this time the patient may be under careful observation and no suspicion be aroused of the existence of suppuration. Then severe headache, vomiting, fever, set in, perhaps with a chill. An Arab was admitted to my wards at the University Hospital in a condition of profound anæmia, having been picked up by the police in the street, covered with blood. There was a small localized area of dulness in the third and fourth interspaces on the right side close to the

sternum, and although no tubercle bacilli were found, it was thought to be probably a localized tuberculosis. He recovered rapidly from the anæmia, and within three months was strong and well. A few days before his intended discharge he began to complain of headache, which became aggravated. He had vomiting, fever, and gradually increasing coma. A large, solitary encapsulated abscess was found in the parieto-occipital region of the left hemisphere, and in the middle lobe of the right lung a circumscribed cavity, probably bronchiectatic, surrounded by fibroid tissue and containing a very offensive pus. So, too, after a blow upon the head or a fracture the symptoms of the lesion may be transient, and months afterward cerebral symptoms of the most aggravated character may develop.

The localization of the lesion is often difficult. In or near the motor region there may be convulsions or paralysis, and it is to be remembered that an abscess in the temporo-sphenoidal lobe may compress the lower motor centres and produce paralysis of the arm and face and on the left side cause aphasia. A large abscess may exist in the frontal lobe without causing paralysis, but in these cases there is almost always some mental dulness. In the temporo-sphenoidal lobe, the common seat, there may be no localizing symptoms. So also in the parieto-occipital region; though here early examination may lead to the detection of hemianopia. In abscess of the cerebellum vomiting is common. If the middle lobe is affected there may be staggering—cerebellar incoördination. Localizing symptoms in the pons and other parts are still more uncertain.

Diagnosis.—In the acute cases there is rarely any doubt. The history of injury followed by fever, marked cerebral symptoms, the development of optic neuritis and rigors, delirium, and perhaps paralysis, make the diagnosis certain. In chronic ear-disease, such cerebral symptoms as drowsiness and torpor, with irregular fever, supervening upon the cessation of a discharge should excite the suspicion of abscess. It is particularly in the chronic cases that difficulties arise. The symptoms resemble those of tumor of the brain; indeed, they are those of tumor plus fever. In a patient with a history of trauma or with localized lung or pleural trouble, who for weeks or months has had slight headache or dizziness, the onset of a rapid fever, intense headache, and vomiting point strongly to abscess.

It is not always easy to determine whether the meninges are involved with the abscess. Often in ear-disease the condition is that of meningo-encephalitis. I have already referred to a condition sometimes associated with ear-disease, which may simulate closely cerebral meningitis or even abscess. Indeed, Gowers states that not only may these general symptoms be produced by ear-disease, but even distinct optic neuritis.

Treatment.—A remarkable advance has been made of late years in dealing with these cases, owing to the impunity with which the brain can be explored. In ear-disease free discharge of the inflammatory products should be promoted and careful disinfection practised. The treatment of injuries and fractures comes within the scope of the surgeon. The acute

symptoms, such as fever, headache, and delirium, must be treated by rest, an ice-cap, and, if necessary, local depletion. In all cases, when a reasonable suspicion exists of the occurrence of abscess, the trephine should be applied and the brain explored. The cases following ear-disease, in which the suppuration is in the temporo-sphenoidal lobe or in the cerebellum, offer the most favorable chances of recovery. The localization can rarely be made accurately in these cases, and the operator must be guided more by general anatomical and pathological knowledge. In cases of injury the trephine should be applied over the seat of the blow or the fracture. In ear-disease the suppuration is most frequent in the temporo-sphenoidal or in the cerebellum, and the operation should be performed at the points most accessible to these regions.

IV. HEMIPLEGIA AND DIPLEGIA IN CHILDREN.

It is as yet hard to say, without fuller knowledge of the etiology of these common conditions, where they should be classified. In a majority of the cases, whatever the nature of the primary pathological change, the final state is one of a chronic encephalitis, often with great atrophy of the convolutions or the formation of large cyst-like spaces—porencephalus.

I. Hemiplegia.

Etiology.—Of 135 cases, comprising those from the Infirmary for Diseases of the Nervous System, Philadelphia, from the Elwyn Institution for Feeble-minded Children, under Kerlin, and from my clinic at the Johns Hopkins Hospital, 60 were in boys and 75 in girls. Right hemiplegia occurred in 79, left in 56. In 15 cases the condition was said to be congenital.

In a great majority the disease sets in during the first or second year; thus of the total number of cases, 95 were under two. Cases above the fifth year are rare, only 10 in my series. Neither alcoholism nor syphilis in the parents appears to play an important *rôle* in this affection. Difficult or abnormal labor is responsible for certain of the cases, particularly injury with the *forceps*. Trauma, such as falls or puncturing wounds, is more rare. The condition followed ligation of the common carotid in one case.

Infectious diseases. All the authors lay special stress upon this factor. In 19 cases in my series the disease came on during or just after one of the specific fevers. I saw one case in which during the height of vaccination convulsions developed, followed by hemiplegia. In a great majority of the cases the disease sets in with a convulsion, in which the child may remain for several hours or longer, and after recovery the paralysis is noticed.

Morbid Anatomy.—In an analysis which I have made of 90 autopsies reported in the literature, the lesions may be grouped under three headings:

(*a*) Embolism, thrombosis, and hæmorrhage, comprising 16 cases, in 7 of which there was blocking of a Sylvian artery, and in 9 hæmorrhage. A striking feature in this group is the advanced age of onset. Ten of the cases occurred in children over six years old.

(*b*) Atrophy and sclerosis, comprising 50 cases. The wasting is either of groups of convolutions, an entire lobe, or the whole hemisphere. The meninges are usually closely adherent over the affected region, though sometimes they look normal. The convolutions are atrophied, firm, and hard, contrasting strongly with the normal gyri. The sclerosis may be diffuse and wide-spread over a hemisphere, or there may be nodular projections—the hypertrophic sclerosis. Some of the cases show remarkable unilateral atrophy of the hemisphere. In one of my cases the atrophied hemisphere weighed 169 grammes and the normal 653 grammes. The brain tissue may be a mere shell over a dilated ventricle.

(*c*) Porencephalus, which was present in 24 of the 90 autopsies. This term was applied by Heschel to a loss of substance in the form of cavities and cysts at the surface of the brain, either opening into and bounded by the arachnoid, and even passing deeply into the hemisphere, or reaching to the ventricle. In the study by Audrey of 103 cases of porencephalus, hemiplegia was mentioned in 68 cases.

Practically, then, in infantile hemiplegia cortical sclerosis and porencephalus are the important anatomical conditions. The primary change in the majority of these cases is still unknown. Porencephalia may result from a defect in development or from hæmorrhage at birth. The etiology is clear in the limited number of cases of hæmorrhage, embolism, and thrombosis, but there remains the large group in which the final change is sclerosis and atrophy. What is the primary lesion in these instances? The clinical history shows that in nearly all these cases the onset is sudden, with convulsions—often with slight fever. Strümpell believes that this condition is due to an inflammation of the gray matter—polio-encephalitis—a view which has not been very widely accepted, as the anatomical proofs are wanting. Gowers suggests that thrombosis may be present in some instances. This might probably account for the final condition of sclerosis, but clinically thrombosis of the veins rarely occurs in healthy children, which appear to be those most frequently attacked by infantile hemiplegia, and post-mortem proof is yet wanting of the association of thrombosis with the disease.

Symptoms.—(*a*) The onset. The disease may set in suddenly without spasms or loss of consciousness. In more than half the cases the child is attacked with partial or general convulsions and loss of consciousness, which may last from a few hours to many days. This is one of the most striking features in the disease. Fever is usually present. The

hemiplegia, noticed as the child recovers consciousness, is generally complete. Sometimes the paralysis is not complete at first, but develops after subsequent convulsions. The right side is more frequently affected than the left. The face is commonly not involved.

(*b*) Residual symptoms. In some cases the paralysis gradually disappears and leaves scarcely a trace as the child grows up. The leg, as a rule, recovers more rapidly and more fully than the arm, and the paralysis may be scarcely noticeable. In a majority of cases, however, there is a characteristic hemiplegic gait. The paralysis is most marked in the arm, which is usually wasted; the forearm is flexed at right angles, the hand is flexed, and the fingers are contracted. Motion may be almost completely lost; in other instances the arm can be lifted above the head. Late rigidity, which almost always develops, is the symptom which suggested the name *hemiplegia spastica cerebralis* to Heine, the orthopædic surgeon who first accurately described these cases. It is, however, not constant. The limbs may be quite relaxed even years after the onset. The reflexes are usually increased. In several instances, however, I have known them to be absent. Sensation is, as a rule, not disturbed.

Aphasia is a not uncommon symptom, and occurred in 16 cases of my series—a smaller number than given in the series of Wallenberg, Gaudard, and Sachs.

Mental Defects.—One of the most serious consequences of infantile hemiplegia is the failure of mental development. A considerable number of these cases drift into the institutions for feeble-minded children. Three grades may be distinguished—idiocy, which is most common when the hemiplegia has existed from birth; imbecility, which often increases with the development of epilepsy; and feeble-mindedness, a retarded rather than an arrested development.

Epilepsy.—Of the cases in my series, 41 were subjects of convulsive seizures, which is one of the most distressing sequences of the disease. The seizures may be either transient attacks of *petit mal*, true Jacksonian fits, beginning in and confined to the affected side, or general convulsions.

Post-hemiplegic Movements.—It was in cases of this sort that Weir Mitchell first described the post-hemiplegic movements. They are extremely common, and were present in 34 of my series. There may be either slight tremor in the affected muscles, or incoördinate choreiform movements—the so-called post-hemiplegic chorea—or, lastly,

Athetosis.—In this condition, described by Hammond, there are remarkable spasms of the paralyzed extremities, chiefly of the fingers and toes, and in rare instances of the muscles of the mouth. The movements are involuntary and somewhat rhythmical; in the hand, movements of adduction or abduction and of supination and pronation follow each other in orderly sequence. There may be hyperextension of the fingers, during which they are spread wide apart. This condition is much more frequent in children than in adults. In the latter it may be combined

with hemianæsthesia, and the lesion is not cortical, but basic in the neighborhood of the thalamus. The movements are sometimes increased by emotion. They usually persist during sleep.

II. Spastic Diplegia—Birth Palsies.

In this condition there is a paralysis with spasm of all extremities, dating from or shortly succeeding birth, more rarely following the fevers or an attack of convulsions. The legs are usually more involved than the arms; there is no wasting, no disturbance of sensation. The reflexes are increased. The mental condition is profoundly disturbed. The patients are usually imbeciles or idiots, helpless in mind and body. Ataxic and athetoid movements of the most exaggerated kind may occur.

While a limited number only of cases of infantile hemiplegia are congenital, on the other hand, in spastic diplegia a large proportion of the cases results from injury at birth. Practically the spastic paraplegia of children should be considered with this condition, as its etiology is essentially the same. The arms, too, may be so slightly affected as to make it difficult to determine whether it is a case of diplegia or paraplegia. The cases usually date from birth, and a majority are born in first labors or are forceps cases. Ross suggests that in feet presentation there may be laceration or tearing of the cerebro-spinal membranes.

Morbid Anatomy.—The birth palsies which ultimately induce the spastic diplegias or paraplegias are most frequently the result of meningeal hæmorrhage. The importance of this condition has been shown by the studies of Litzmann and Sarah J. McNutt. The bleeding may come from the veins, or, in one case which I saw with Hirst, from the longitudinal sinus. The bleeding has in many cases been thickest over the motor areas, and it seems probable that the sclerosis found in these cases may result from the compression of the blood-clot. In other instances the condition may be due to a fœtal meningo-encephalitis. In sixteen autopsies collected in the literature, in which the patients died at ages varying from two to thirty, the anatomical condition was either a diffuse atrophy, which was most common, or porencephalus.

Symptoms.—At first nothing abnormal may be noticed about the child. In some instances there have been early and frequent convulsions; then at the age when the child should begin to walk it is noticed that the limbs are not used readily, and on examination a stiffness of the legs and arms is found. Even at the age of two the child may not be able to sit up, and often the head is not well supported by the neck muscles. The rigidity, as a rule, is more marked in the legs, and there is adductor spasm. When supported on the feet, the child either rests on its toes and the inner surface of the feet, with the knees close together, or the legs may be crossed. The stiffness of the upper limbs varies. It may be scarcely noticeable or the rigidity may be as marked as in the legs. Con-

stant irregular movements of the arms are not uncommon. The child has great difficulty in grasping an object. The spasm and weakness may be more evident on one side than the other. The mental condition is, as a rule, defective and convulsive seizures are common.

Associated with the spastic paraplegia are two allied conditions of considerable interest, characterized by spasm and disordered movements. A child with spastic diplegia may present, in an unusual degree, irregular movements of the muscles. In attempting to grasp an object the fingers may be thrown out in a stiff, spasmodic, irregular manner, or there may be constant irregular movements of the shoulders, arms, and hands, with slight incoördination of the head. Cases of this description have been described as *chorea spastica*, and they may be difficult to separate from multiple sclerosis and from Friedreich's ataxia.

A still more remarkable condition is that of *bilateral athetosis*, in which there is a combination of spasm more or less marked with the most extraordinary bizarre movements of the muscles. The condition, as a rule, dates from infancy. The patient may not be able to walk. The head is turned from side to side; there are continual irregular movements of the face muscles, and the mouth is drawn and greatly distorted. The extremities are more or less rigid, particularly in extension. On making the slightest attempt to move, often spontaneously, there are extraordinary movements of the arms and legs, particularly of the arms, somewhat like though much more exaggerated than athetosis. The patients are often unable to help themselves on account of these movements. The reflexes are increased. The mental condition is variable. The patient may be idiotic, but in two of the four cases which I have seen the patients were intelligent. Massalongo,* who has carefully studied this condition, describes three cases in one family. I have collected fifty-three cases from the literature, thirty-three of which occurred in males and twenty in females. There have been three autopsies. In Kurella's case there was pachymeningitis and bilateral lesions of the motor convolutions. Déjérine's patient had atrophy of the convolutions on both sides, while in my case the brain macroscopically presented no changes.

III. Spastic Paraplegia.

This condition, which is more fully described under the section upon the spinal cord, is in reality a cerebral affection, and may be due to conditions similar to those found in spastic diplegia. Indeed, it may at first be difficult to determine whether the arms are involved or not. The evidence of the cerebral origin of the affection is based upon the frequent coexistence of idiocy, imbecility, and nystagmus, and the occurrence of cases of spastic diplegia, in which the paraplegic symptoms are identical. All grades are met with, from pure spastic paraplegia with perfect use of the

* Dell' Atetosi Doppia, Collezione Italiana di Letture sulla Medicina, Series V, N. 3

arms to the most extreme bilateral spasm. There have been, so far as I know, only two autopsies in this disease: the case of Förster, in which there was a moderate grade of general cortical sclerosis with slight dilatation of the ventricles, and the recent case of Sachs, in which there was a meningo-encephalitis with atrophy and descending degeneration of both lateral columns.

Treatment.—The possibility of injury to the brain in protracted labor and in forceps cases should be borne in mind by the practitioner. The former entails the greatest risk. In infantile hemiplegia the physician at the outset sees a case of ordinary convulsions, perhaps more protracted and severe than usual. These should be checked as rapidly as possible by the use of the bromides, the application of cold or heat, and a brisk purge. During convulsions chloroform may be administered with safety even to the youngest children. When the paralysis is established not much can be hoped from medicines. In only rare instances does the paralysis entirely disappear. The indications are to favor the natural tendency to improve by maintaining the general nutrition of the child, to lessen the rigidity and contractures by massage and passive motion, and if necessary to correct deformities by mechanical or surgical measures. Much may be done by careful manipulation and rubbing and the application of a proper apparatus. In children the aphasia usually disappears. The epilepsy is a distressing and obstinate symptom, for which a cure can rarely be anticipated. Prolonged periods of quiescence are, however, not uncommon. In the Jacksonian fits the bromides rarely do good, unless there is much irritability and excitement. Operative measures, which have been carried out in several cases, have not been successful. The liability to feeble-mindedness is the most serious outlook in the infantile cerebral palsies. In many cases the damage is irreparable, and idiocy and imbecility result. With patient training and with care many of the children reach a fair measure of intelligence and self-reliance.

V. SCLEROSIS OF THE BRAIN.

General Remarks.—The connective tissue of the central nervous system is of two kinds—one, the neuroglia, special and peculiar, derived from the ectoderm, with distinct morphological and chemical characters; the other, derived from the mesoderm, identical with the ordinary collagenous fibrous tissue of the body. Both play important parts in indurative processes in the brain and cord. A convenient division of the cerebro-spinal scleroses is into degenerative, inflammatory, and developmental forms.

The *degenerative scleroses* comprise the largest and most important subdivision, in which provisionally the following groups may be made: (*a*) The common secondary degeneration which follows when nerve-fibres

are cut off from their trophic centres; (*b*) toxic forms, among which may be placed the scleroses from lead and ergot, and, most important of all, the sclerosis of the posterior column, due in such a large proportion of cases to the virus of syphilis. Other unknown toxic agents may possibly induce degeneration of the nerve-fibres in certain tracts. The systemic paths in the cord differ apparently in their susceptibility and the posterior columns appear most prone to undergo this change; (*c*) the sclerosis associated with change in the smaller arteries and capillaries, which is met with as a senile process in the convolutions. In all probability some of the forms of insular sclerosis are due to primary alterations in the blood-vessels; but it is not yet settled whether the lesion in these cases is a primary degeneration of the nerve cells and fibres to which the sclerosis is secondary, or whether the essential factor is an alteration in nutrition caused by lesions of the capillaries and smaller arteries.

The *inflammatory scleroses* embrace a less important and less extensive group, comprising secondary forms which develop in consequence of irritative inflammation about tumors, foreign bodies, hæmorrhages, and abscess. Histologically these are chiefly mesodermic (vascular) scleroses, which arise from the connective tissue about the blood-vessels. Possibly a similar change may follow the primary, acute encephalitis, which Strümpell holds is the initial lesion in the cortical sclerosis which is so commonly found post mortem in infantile hemiplegia.

The *developmental scleroses* are believed to be of a purely neurogliar character, and embrace the new growth about the central canal in syringomyelia and, according to recent French writers, the sclerosis of the posterior columns in Friedreich's ataxia. It is stated that histologically this form is different from the ordinary variety. It may be, too, that the diffuse cortical sclerosis met with as a congenital condition without thickening of the meninges belongs to this type. It is not improbable that many forms of sclerosis are of a mixed character, in which both the ectodermic and mesodermic connective tissues are involved.

Anatomically we meet with the following varieties:

(1) **Miliary sclerosis** is a term which has been applied to several different conditions. Gowers mentions a case in which there were grayish-red spots at the junction of the white and gray matters, and in which the neuroglia was increased. There is also a condition in which, on the surface of the convolutions, there are small nodular projections, varying from a half to five or more millimetres in diameter. Single nodules of this sort are not uncommon; sometimes they are abundant. So far as is known no symptoms are produced by them.

(2) **Diffuse sclerosis,** which may involve an entire hemisphere, or a single lobe, in which case the term *sclérose lobaire* has been applied to it by the French. It is not an important condition in general medical practice, but occurs most frequently in idiots and imbeciles. In extensive

cortical sclerosis of one hemisphere the ventricle is usually dilated.* The symptoms of this condition depend upon the region affected. There may be a considerable extent of sclerosis without symptoms or without much mental impairment. In a majority of cases there is hemiplegia or diplegia with imbecility or idiocy.

(3) **Tuberous Sclerosis.**—In this remarkable form, which is also known as hypertrophic, there are on the convolutions areas projecting beyond the surface of an opaque white color and exceedingly firm. The sclerosis may not disturb the symmetry of the convolution, but simply cause a great enlargement, increase in the density, and a change in the color.

These three forms are not of much practical interest except in asylum and institution work. The last variety forms a well-characterized disease of considerable importance, namely:

(4) Insular Sclerosis (*Sclérose en plaques*).

Definition.—A chronic affection of the brain and cord, characterized by localized areas in which the nerve elements are more or less replaced by connective tissue. This may occur in the brain or cord alone, more commonly in both.

Etiology.—This is obscure. Kahler, Marie, and others assign great importance to the infectious diseases, particularly scarlet fever. It is found most commonly in middle-aged persons, but cases are not uncommon in children, in whom Pritchard states that more than fifty cases have been reported.†

Morbid Anatomy.—The sclerotic areas are widely distributed through the brain and cord. Cases limited to the cord are almost unknown. On section of the brain, grayish-red areas are seen scattered through the white matter. The cortex is not often involved. The patches are sometimes abundant in the neighborhood of the ventricles, and in the pons, cerebellum, basal ganglia, and the medulla. The cord may be only slightly involved or there may be irregular areas in different regions. Histologically in the sclerosed patches there is great increase in the connective tissue, the fibres of which are denser and firmer. The gradual growth destroys the medulla of the nerves, but the axis cylinders persist in a remarkable way.

Symptoms.—The onset is slow and the disease is chronic. Feebleness of the legs with irregular pains and stiffness are among the early symptoms. Indeed, the clinical picture may be that of spastic paraplegia with great increase in the reflexes. The following are the most important features:

(a) *Volitional Tremor.*—There is no paralysis of the arms, but on at-

* In my monograph on Cerebral Palsies of Children I have given a description of the distribution of the sclerosis in ten specimens in the museum at the Elwyn Institution.

† Cyclopedia of the Diseases of Children, vol. iv.

tempting to pick up an object there is trembling or rapid oscillation. A patient may be unable to lift even a glass of water to the mouth. The tremor may be marked in the legs and in the head, which shakes as he walks. When the patient is recumbent the muscles may be perfectly quiet. On attempting to raise the head from the pillow, trembling at once comes on. (*b*) *Scanning Speech.*—The words are pronounced slowly and separately, or the individual syllables may be accentuated. This staccato or syllabic utterance is a common feature. (*c*) Nystagmus, a rapid oscillatory movement of both eyes, constitutes an important symptom.

Sensation is unaffected in a majority of the cases. Optic atrophy sometimes occurs, but not so frequently as in tabes. The sphincters, as a rule, are unaffected until the last stages. Mental debility is not uncommon. Remarkable remissions occur in the course of the disease, in which for a time all the symptoms may improve. Vertigo is common, and there may be sudden attacks of coma, such as occur in general paresis.

The *diagnosis* in well-marked cases is easy. Volitional tremor, scanning speech, and nystagmus form a characteristic symptom-group. With this there is usually more or less spastic weakness of the legs. Paralysis agitans, certain cases of general paresis, and occasionally hysteria may simulate the disease very closely. If the case is not seen until near the end the diagnosis may be impossible. Buzzard holds that of all organic diseases of the nervous system disseminated sclerosis in its early stages is that which is most commonly mistaken for hysteria.

Much more puzzling, however, are the instances of *pseudo-sclérose en plaques*, which have been described by Westphal. The volitional tremor, the scanning speech, and the spastic condition are present, but no lesions have been found post mortem. The movements in this form are more violent, but nystagmus does not occur. Some of the cases may possibly be examples of general paresis. In children the condition may with difficulty be separated from Friedreich's ataxia.

The *prognosis* is unfavorable. Ultimately, the patient, if not carried off by some intercurrent affection, becomes bedridden.

Treatment.—No known treatment has any influence on the progress of sclerosis of the brain. Neither the iodides nor mercury have the slightest effect, but a prolonged course of nitrate of silver may be tried.

VI. CHRONIC DIFFUSE MENINGO-ENCEPHALITIS

(Dementia Paralytica; General Paresis).

Definition.—A chronic, progressive meningo-encephalitis associated with psychical and motor disturbances, finally leading to dementia and paralysis.

Etiology.—Males are affected much more frequently than females. It occurs chiefly between the ages of thirty and fifty-five. Heredity is a

factor in only a few cases. An overwhelming majority of the cases are in married people. Statistics show that it is more common in the lower classes of society, but in this country in general medical practice the disease is certainly more common in the well-to-do classes. An important predisposing cause is "a life absorbed in ambitious projects with all its strongest mental efforts, its long-sustained anxieties, deferred hopes, and straining expectation" (Mickle). The habits of life so frequently seen in active business men in our large cities, and well expressed by the phrase "burning the candle at both ends," strongly predispose to the disease. Among other factors of importance are syphilis, excesses *in baccho et venere*, injuries, and chronic lead-poisoning.

Morbid Anatomy.—The essential histological changes in the cerebral cortex are thus summarized by Bevan Lewis: (1) A stage of inflammatory change in the tunica adventitia of the arteries with excessive nuclear proliferation, profound changes in the vascular channels, and trophic changes induced in the tissues around.

(2) A stage of extraordinary development of the lymph-connective system of the brain, with a parallel degeneration and disappearance of nerve elements and the axis cylinders of which they are denuded.

(3) A stage of general fibrillation with shrinking and extreme atrophy of the parts involved.

The macroscopical changes are: (*a*) Increase in the cerebro-spinal fluid, œdema of the pia, and thickening and opacity of the meninges, which are adherent in places and tear the cortex on removal. The dura is sometimes thickened, and pachymeningitis hæmorrhagica interna may be present.

(*b*) The convolutions are atrophied, usually in a marked degree, and in consequence the brain looks small. This is particularly noticeable in the frontal and parietal regions. On section it cuts with firmness. In extreme cases the gray matter may be obscurely outlined. The grade of sclerosis varies much in different cases. The white matter may be firmer in consistence, but it does not show such important changes. The ventricles are dilated and the ependyma extremely granular. In addition, there are frequently areas of softening or hæmorrhage associated with chronic arterio-sclerosis.

(*c*) Spinal cord. Changes occur leading to increase in the connective-tissue elements and frequently to degeneration of the pyramidal tracts secondary to the cortical lesion. The posterior columns may also be involved, and occasionally the distribution of the sclerosis is that of the amyotrophic form.

Symptoms.—(*a*) *Prodromal Stage.*—This is of variable duration, and is characterized by a general mental state which finds expression in symptoms trivial in themselves but important in connection with others. Irritability, inattention to business amounting sometimes to indifference or apathy, and sometimes a change in character marked by acts which

may astonish the friends and relatives may be the first indications. Instead of apathy or indifference there may be an extraordinary degree of physical and mental restlessness. The patient is continually planning and scheming, or may launch into extravagances and speculation of the wildest character. A common feature at this period is the display of an unbounded egoism. He boasts of his personal attainments, his property, his position in life, or of his wife and children. Following these features are important indications of moral perversion, manifested in offences against decency or the law, many of which acts have about them a suspicious effrontery. Forgetfulness is common, and may be shown in inattention to business details and in the minor courtesies of life. At this period there may be no motor phenomena. The onset of the disease is usually insidious, although cases are reported in which epileptiform or apoplectiform seizures were the first symptoms. Among the early motor features are tremor of the tongue and lips in speaking, slowness of speech and hesitancy, and inequality of the pupils.

(*b*) *Second Stage.*—This is characterized in brief by mental exaltation or excitement and a progress in the motor symptoms. "The intensity of the excitement is often extreme, acute maniacal states are frequent; incessant restlessness, obstinate sleeplessness, noisy, boisterous excitement, and blind, uncalculating violence especially characterize such states" (Lewis). It is at this stage that the delusion of grandeur becomes marked and the patient believes himself to be possessed of countless millions or to have reached the most exalted sphere possible in profession or occupation. This expansive delirium, as it is called, is, however, not characteristic, as was formerly supposed, of paralytic dementia. Besides, it does not always occur, but in its stead there may be marked melancholia or hypochondriasis, or, in other instances, alternate attacks of delirium and depression.

The facies has a peculiar stolidity, and in speaking there is marked tremulousness of the lips and facial muscles. The tongue is also tremulous, and may be protruded with difficulty. The speech is slow, interrupted, and blurred. Writing becomes difficult on account of unsteadiness of the hand. The subject matter of the patient's letters give valuable indications of the mental condition. In many instances the pupils are unequal, irregular, sluggish, sometimes large. Important symptoms in this stage are apoplectiform seizures and paralysis. There may be slight syncopal attacks in which the patient turns pale and may fall. Some of these are *petit mal*. In the true apoplectiform seizure the patient falls suddenly, becomes unconscious, the limbs are relaxed, the face is flushed, the breathing stertorous, the temperature increased, and death may occur. The epileptic seizures are more common than the apoplectiform and may occur early in the disease. A definite aura is not uncommon. The attack usually begins on one side and may not spread. There may be twitchings either in the facial or brachial muscles. Typical Jack-

sonian epilepsy may occur. In a case which died recently under my care, these seizures were among the early symptoms and the disease was regarded as cerebral syphilis. Paralysis, either monoplegic or hemiplegic, may follow these epileptic seizures, or may come on with great suddenness and be transient. In this stage the gait becomes impaired, the patient trips readily, has difficulty in going up or down stairs, and the walk may be spastic or occasionally tabetic. This paresis may be progressive. The knee-jerk is usually increased. Bladder or rectal symptoms gradually develop. The patient becomes helpless, bedridden, and completely demented, and unless care is taken may suffer from bedsores. Death occurs from exhaustion or from some intercurrent affection.

Diagnosis.—The recognition of the disease in the earliest stage is extremely difficult, as it is often impossible to decide that the slight alteration in conduct is anything more than one of the moods or phases to which most men are at times subject. The following description by Folsom is an admirable presentation of the diagnostic characters of the early stage of the disease: "It should arouse suspicion if, for instance, a strong, healthy man, in or near the prime of life, distinctly not of the 'nervous,' neurotic, or neurasthenic type, shows some loss of interest in his affairs or impaired faculty of attending to them; if he becomes varyingly absent-minded, heedless, indifferent, negligent, apathetic, inconsiderate, and, although able to follow his routine duties, his ability to take up new work is, no matter how little, diminished; if he can less well command mental attention and concentration, conception, perception, reflection, judgment; if there is an unwonted lack of initiative, and if exertion causes unwonted mental and physical fatigue; if the emotions are intensified and easily change, or are excited readily from trifling causes; if the sexual instinct is not reasonably controlled; if the finer feelings are even slightly blunted; if the person in question regards with a placid apathy his own acts of indifference and irritability and their consequences, and especially if at times he sees himself in his true light and suddenly fails again to do so; if any symptoms of cerebral vaso-motor disturbances are noticed, however vague or variable."

There are cases of cerebral syphilis which closely simulate dementia paralytica. The mode of onset is important, particularly since paralytic symptoms are usually early in syphilis. The affection of the speech and tongue is not present. Epileptic seizures are more common and more liable to be cortical or Jacksonian in character. The expansive delirium is rare. While symptoms of general paresis are not common in connection with the development of gummata or definite gummatous meningitis, there are, on the other hand, instances of paresis which follow syphilitic infection so closely that an etiological connection between the two must be acknowledged. Post mortem in such cases there may be nothing more than a general arterio-sclerosis and diffuse meningo-encephalitis, which may present nothing distinctive, but the lesions, nevertheless, may be caused by

the syphilitic virus. There are certain forms of lead encephalopathy which resemble general paresis, and, considering the association of plumbism with arterio-sclerosis, it is not unlikely that the anatomical substratum of the disease may result from this poison.

Prognosis.—The disease rarely ends in recovery. As a rule the progress is slowly downward and the case terminates in a few years, although it is occasionally prolonged ten or fifteen years.

Treatment.—The only hope of permanent relief is in the cases following syphilis, which should be placed upon large doses of iodide of potassium. Careful nursing and the orderly life of an asylum are the only measures necessary in a great majority of the cases. For sleeplessness and the epileptic seizures bromides may be used. Prolonged remissions, which are not uncommon, are often erroneously attributed to the action of remedies.

VII. TUMORS OF THE BRAIN.

The following are the most common varieties of new growths within the cranium:

(*a*) *Tubercle*, which may form small or large growths, usually multiple. They are most frequent early in life. Three fourths of the cases occur under twenty, and one half of the patients are under ten years of age (Gowers). Of 299 cases of tumor in persons under nineteen collected from various sources by Starr, 152 were tubercle. They are most numerous in the cerebellum and about the base.

(*b*) *Syphiloma* is most commonly found in the hemispheres or about the pons. The tumors are superficial, attached to the arteries or the meninges, and rarely grow to a large size. They may be multiple.

(*c*) *Glioma and Neuroglioma.*—These vary greatly in appearance. They may be firm and hard, almost like an area of sclerosis, or soft and very vascular. They persist remarkably for many years. Klebs has called attention to the occurrence of elements in them not unlike ganglion-cells. Tumors of this character contain "the spinnen" or spider cells; enormous spindle-shaped cells with single large nuclei; cells like the ganglion-cells of nerve-centres with nuclei and one or more processes; and translucent, band-like fibres, tapering at each end, which result from a vitreous or hyaline transformation of the large spindle-cells.

(*d*) *Sarcoma* occurs most commonly in the membranes of the brain and in the pons. It forms some of the largest and most diffusely infiltrating of intracranial growths.

(*e*) *Carcinoma* not infrequently is secondary to cancer in other parts. It is seldom primary. Occasionally cancerous tumors have been found in symmetrical parts of the brain.

(*f*) Other varieties occur, such as fibroid growths, which usually develop from the membranes; bony tumors, which grow sometimes from

the falx, and psammoma and cholesteatoma. Fatty tumors are occasionally found on the corpus callosum.

(*g*) *Cysts* occur between the membranes and the brain, the result of hæmorrhage or of softening. Porencephalus is a sequence of congenital atrophy or of hæmorrhage, or may be due to a developmental defect. Hydatid cysts will be referred to in the section on parasites.

Symptoms.—(1) **General.**—The following are the most important: *Headache*, either dull, aching, and continuous, or sharp, stabbing, and paroxysmal. It may be diffused over the entire head or limited to the back or front. In the former case it may extend down the neck, and in the latter be accompanied with neuralgic pains in the face. Occasionally the pain may be very localized and associated with tenderness on pressure.

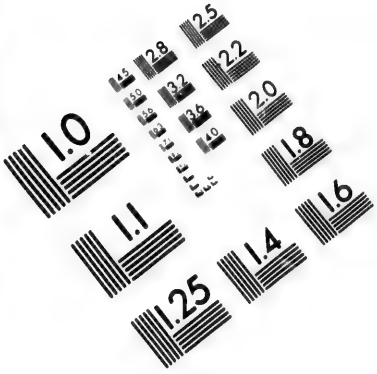
Optic Neuritis.—This occurs in four fifths of all the cases (Gowers). It is usually double, but occasionally is found in only one eye. A growth may develop slowly and attain considerable size without producing optic neuritis. On the other hand, it may occur with a very small tumor, more commonly in a growth at the base.

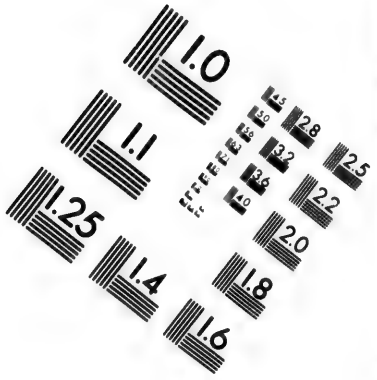
Vomiting.—This is a common feature, and with headache and optic neuritis makes up the characteristic symptom group of cerebral tumor. An important point is the absence of definite relation to the meals. It may be very obstinate, particularly in growths of the cerebellum and the pons.

Giddiness.—This is often an early symptom. The patient complains of vertigo on rising suddenly or on turning quickly. *Mental Disturbance.* The patient may act in an odd, unnatural manner, or there may be stupor and heaviness. The patient may become emotional or silly, or symptoms resembling hysteria may develop. *Convulsions*, either general and resembling true epilepsy or localized (Jacksonian) in character.

(2) **Localizing Symptoms.**—(*a*) *Central Motor Area.*—The symptoms are either irritative or destructive in character. Irritation in the lower third may produce spasm in the muscles of the face, in the angle of the mouth, or in the tongue. The spasm with tingling may be strictly limited to one muscle group before extending to others, and this Seguin terms the *signal symptom.* The middle third of the motor area contains the centres controlling the arm, and here, too, the spasm may begin in the fingers, in the thumb, in the muscles of the wrist, or in the shoulder. In the upper third of the motor areas the irritation may produce spasm beginning in the toes, in the ankles, or in the muscles of the leg. In many instances the patient can determine accurately the point of origin of the spasm, and there are important sensory disturbances, such as numbness and tingling, which may be felt first at the region affected.

In all cases it is important to determine, first, the point of origin, the *signal symptom;* second, the order or march of the spasm; and third, the subsequent condition of the parts first affected, whether it is a state of paresis or anæsthesia.





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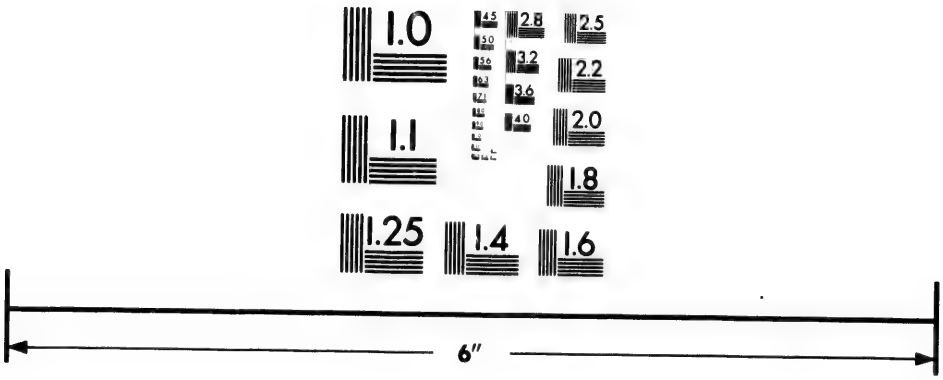

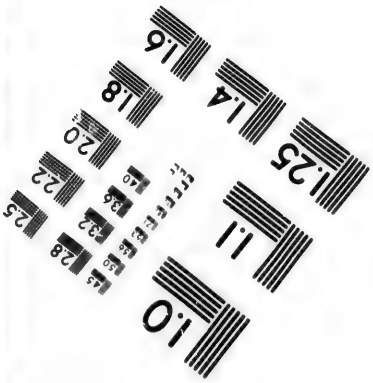

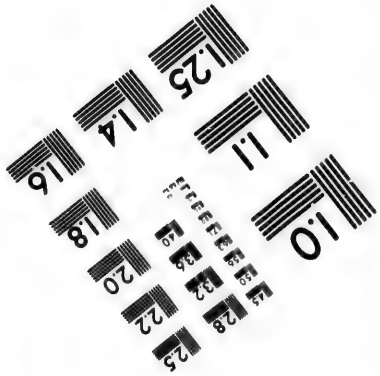

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Destructive lesions in the motor zone cause paralysis, which is often preceded by local convulsive seizures; there may be a monoplegia, as of the leg, and convulsive seizures in the arm, often due to irritation in these centres. Tumors in the neighborhood of the motor area may cause localized spasms and subsequently, as the centres are invaded by the growth, paralysis occurs. On the left side, growths in the third frontal or Broca's convolution may cause motor aphasia.

(*b*) *Prefrontal Region.*—Neither motor nor sensory disturbance may be present. The general symptoms are often well marked. The most striking feature of growths in this region is mental torpor and gradual imbecility. In its extension downward the tumor may involve on the left side the lower frontal convolution and produce aphasia, or in its progress backward cause irritative or destructive lesions of the motor area.

(*c*) Tumors in the *parieto-occipital lobe* may grow to large size without causing any symptoms. There may be word-blindness and mind-blindness when the angular gyrus is involved, and paraphasia.

(*d*) Tumors of the *occipital lobe* produce hemianopia, and a bilateral lesion may produce blindness. Tumors in this region on the left hemisphere may be associated with word-blindness and mind-blindness.

(*e*) Tumors in the *temporal lobe* may attain a large size without producing symptoms. In their growth they involve the lower motor centres. On the left side involvement of the first and second gyri may be associated with word-deafness.

(*f*) Tumors growing in the neighborhood of the *basal ganglia* produce hemiplegia from involvement of the internal capsule. Limited growths in either nucleus of the corpus striatum do not necessarily cause paralysis. Tumors in the thalamus opticus may also, when small, cause no symptoms, but increasing they may involve the fibres of the optic radiation, producing hemianopia and sometimes hemianæsthesia. Growths in this situation are apt to cause early optic neuritis and, growing into the third ventricle, may cause a distention of the lateral ventricles. In fact, pressure symptoms from this cause and paralysis due to involvement of the internal capsule are the chief symptoms of tumor in and about these ganglia.

Growths in the *corpora quadrigemina* are rarely limited, but most commonly involve the crura cerebri as well. Ocular symptoms are marked. The pupil reflex is lost and there is nystagmus. In the gradual growth the third nerve is involved as it passes through the crus, in which case there will be motor oculi paralysis on one side and hemiplegia on the other, a combination almost characteristic of unilateral crus disease.

(*g*) Tumors of the *pons* and *medulla.* The symptoms are chiefly those of pressure upon the nerves emerging in this region. In disease of the pons the nerves may be involved alone or with the tract. Of 52 cases analyzed by Mary Putnam Jacobi, there were 13 in which the cranial nerves were involved alone, 13 in which the limbs were affected, and 26 in which there was hemiplegia and involvement of the nerves. Twenty-two

of the latter had what is known as alternate paralysis—i. e., involvement of the nerves on one side and the limbs on the opposite side. In four cases there were no motor symptoms. A tumor growing in the lower part of the pons usually involves the sixth nerve, producing internal strabismus; the seventh nerve, producing facial paralysis; and the auditory nerve, causing deafness. Conjugate deviation of the eyes to the side opposite that on which there is facial paralysis also occurs.

Tumors of the *medulla* may involve the cranial nerves alone or cause in some instances a combination of hemiplegia with paralysis of the nerves. Signs of irritation in the ninth, tenth, and eleventh nerves are usually present, and produce difficulty in swallowing, irregular action of the heart, irregular respiration, vomiting, and sometimes retraction of the head and neck. The gait may be unsteady or, if there is pressure on the cerebellum, ataxic. Occasionally there are sensory symptoms, numbness, and tingling. Toward the end convulsions may occur.

(*h*) Tumors of the *cerebellum* constitute by far the most important affection of this part. There may be no symptoms whatever if the tumor is confined to one hemisphere and does not involve the middle lobe. When this portion is affected the symptoms are very characteristic, consisting of:

Vertigo, which is more constant in this than in affections of any other region of the brain. This may be due, some believe, to the central relations of the semicircular canals with the cerebellum. The giddiness may be of the most distressing nature.

Headache.—In the analysis by Mary Putnam Jacobi of symptoms in tumors in various parts of the brain headache was relatively much more frequent in tumors of the cerebellum than in any other region.

Cerebellar Ataxia.—The gait is irregular and staggering. In attempting to walk the patient reels to and fro like a drunken man. There may be a tendency to fall to one side, backward, or, less commonly, forward.

Other less constant but suggestive symptoms are the optic neuritis; nystagmus; neuralgic pains in the region of the neck and occiput; pressure symptoms on the medulla, causing vomiting; distention of the lateral ventricles, causing in children hydrocephalus; and, lastly, bilateral rigidity from pressure on the motor paths (Sharkey).

Diagnosis.—From the general symptoms alone the existence of tumor may be determined, for the combination of headache, optic neuritis, and vomiting is distinctive. The localization must be gathered from the consideration of the symptoms above detailed. Mistakes are most likely to occur in connection with uræmia, hysteria, and general paralysis; but careful consideration of all the circumstances of the case usually enables the practitioner to avoid error.

Prognosis.—Syphilitic tumors alone are amenable to treatment. Tuberculous growths occasionally cease to grow and become calcified. The gliomata and fibromata, particularly when the latter grow from the membranes, may last for years. I have described a case of small, hard

glioma, in which the Jacksonian epilepsy persisted for fourteen years. Hughlings Jackson has reported cases of glioma in which the symptoms lasted for over ten years. The more rapidly growing sarcomata usually prove fatal in from six to eighteen months. Death may be sudden, particularly in growths near the medulla; more commonly it is due to coma in consequence of gradual increase in the intracranial pressure.

Treatment.—(*a*) *Medical.*—If there is a suspicion of syphilis the iodide of potassium and mercury should be given. Nowhere do we see more brilliant therapeutical effects than in certain cases of cerebral gummata. The iodide should be given in increasing doses. In tuberculous tumors the outlook is less favorable, though instances of cure are reported, and there is post-mortem evidence to show that the solitary tuberculous tumors may undergo changes and become obsolete. A general tonic treatment is indicated in these cases. The headache usually demands prompt treatment. The iodide of potassium in full doses sometimes gives marked relief. An ice-cap for the head or, in the occipital headache, the application of the Paquelin cautery may be tried. The bromides are not of much use in the headache from this cause, and, as the last resort, morphia must be given. For the convulsions bromide of potassium is of little service.

(*b*) *Surgical.*—Tumors of the brain have been successfully removed by Macewen, Horsley, Keen, and others. The number of cases for operation, however, is small. Four fifths at least of all the cases are probably unsuccessful, or of such a nature as to render an operation fatal. The most advantageous cases are the localized fibromata growing from the dura and only compressing the brain substance, as in Keen's remarkable case. The safety with which the exploratory operation can be made warrants it in all doubtful cases.

VIII. CHRONIC HYDROCEPHALUS.

Definition.—A condition, congenital or acquired, in which there is a great accumulation of fluid within the ventricles of the brain.

The term hydrocephalus has also been applied to the collection of fluid between the cortex of the brain and the skull, known in this situation as *h. externus* or *h. ex vacuo*, a condition common in cases of atrophy of the brain substance, and perhaps caused also by meningeal cysts. A true dropsy, however, of the arachnoid sac probably does not occur.

The cases may be divided into two groups, congenital or infantile, and secondary or acquired.

(1) **Congenital Hydrocephalus.**—The enlarged head may obstruct labor; more frequently the condition is noticed some time after birth. The cause is unknown. It has occurred in several members of the same family.

The anatomical condition in these cases offers no clew to the nature of

the trouble. The lateral ventricles are enormously distended, but the ependyma is usually clear, sometimes a little thickened and granular, and the veins large. The choroid plexuses are vascular, sometimes sclerotic, but often natural-looking. The third ventricle is enlarged, the aqueduct of Sylvius dilated, and the fourth ventricle may be distended. The quantity of fluid may reach several litres. It is limpid and contains a trace of albumen and salts. The changes in consequence of this enormous ventricular distention are remarkable. The cerebral cortex is greatly stretched, and over the middle region the thickness may amount to no more than a few millimetres without a trace of the sulci or convolutions. The basal ganglia are flattened. The skull enlarges, and the circumference of the head of a child of three or four years may reach twenty-five or even thirty inches. The sutures widen, Wormian bones develop in them, and the bones of the cranium become exceedingly thin. The veins are marked beneath the skin. A fluctuation wave may sometimes be obtained, and Fisher's brain murmur may be heard. The orbital plates of the frontal bone are depressed, causing exophthalmos, so that the eyeballs cannot be covered by the eyelids.

Convulsions may occur. The reflexes are increased, the child learns to walk late, and ultimately in severe cases the legs become feeble and sometimes spastic. The mental condition is variable; the child may be bright, but, as a rule, there is some grade of imbecility. The congenital cases usually die within the first four or five years. The process may be arrested and the patient may reach adult life. Cases of this sort are not very uncommon. Even when extreme, the mental faculties may be retained, as in Bright's celebrated patient, Cardinal, who lived to the age of twenty-nine, and whose head was translucent when the sun was shining behind him. Care must be taken not to mistake the rachitic head for hydrocephalus.

(2) **Acquired Chronic Hydrocephalus.**—This is stated to be occasionally primary (idiopathic)—that is to say, it comes on spontaneously in the adult without observable lesion. Dean Swift is said to have died of hydrocephalus, but this seems very unlikely. It is based upon the statement that "he (Mr. Whiteway) opened the skull and found much water in the brain," a condition no doubt of *h. ex vacuo*, due to the wasting associated with his prolonged illness and paralysis. In nearly all cases there is either a tumor at the base of the brain or in the third ventricle, which compresses the venæ Galeni. The passage from the third to the fourth ventricle may be closed, either by a tumor or by parasites. More rarely the foramen of Magendie, through which the ventricles communicate with the cerebrospinal meninges, becomes closed by meningitis. These conditions, occurring in adults, may produce the most extreme hydrocephalus without any enlargement of the head. Even when the tumor begins early in life there may be no expansion of the skull. In the case of a girl aged sixteen, blind from her third year, the head was not unusually large, the ventricles were

enormously distended, and in the Rolandic region the brain substance was only five millimetres in thickness. A tumor occupied the third ventricle. In a case of cholesteatoma of the floor of the third ventricle, in which the symptoms persisted at intervals for eight or nine years, the ventricles were enormously distended without enlargement of the skull. In other instances the sutures separate and the head gradually enlarges.

The symptoms of hydrocephalus in the adult are curiously variable. In the case first mentioned there were early headaches and gradual blindness; then a prolonged period in which she was able to attend to her studies. Headaches again supervened, the gait became irregular and somewhat ataxic. Death occurred suddenly. In the other case there were prolonged attacks of coma with a slow pulse, and on one occasion the patient remained unconscious for more than three months. Gradually progressing optic neuritis without focalizing symptoms, headache, and attacks of somnolence or coma are suggestive symptoms. Cases are rare as a result of meningitis. The only instances I have seen were two which corresponded to the posterior meningitis of Gee and Barlow, in which, with the distention, there was extensive chronic purulent ependymitis.

Treatment.—Very little can be done to relieve hydrocephalus. Medicines are powerless to cause the absorption of the fluid. More rational is the system of gradual compression, with or without the withdrawal of small quantities of the fluid. The compression may be made by means of broad plasters, so applied as to cross each other on the vertex, and another may be placed round the circumference.

Of late years puncture of the ventricles, an operation which had been abandoned, has been revived, particularly by Keen, and in a few cases is justifiable. When pressure symptoms are marked it may be employed with great relief to the headache and removal of the spastic state of the legs. Quincke recommends, and has practised in these cases, as well as in acute hydrocephalus, puncture of the subarachnoid sac between the third and the fourth lumbar vertebræ. At this point the spinal cord cannot be touched. The advantage is a slower removal of fluid and less danger of collapse.

IV. GENERAL AND FUNCTIONAL DISEASES.

I. ACUTE DELIRIUM (*Bell's Mania*).

Definition.—Acute delirium running a rapidly fatal course, with slight fever, and in which post mortem no lesions are found sufficient to account for the disease.

Cases are reported by many old writers under the term brain fever or phrenitis. Bell, at the time Superintendent of the McLean Asylum, de-

scribed it* accurately under the designation, "a form of disease resembling some advanced stages of mania and fever."

The disease may set in abruptly or be preceded by a period of irritability, restlessness, and insomnia. The mental symptoms develop with rapidity and may quickly reach a grade of the most intense frenzy. There are the wildest hallucinations and outbreaks of great violence. The patient talks incessantly, but incoherently and unintelligibly. No sleep is obtained, and at last, worn out with the intensity of the muscular movements, the patient becomes utterly prostrated and assumes the sitting or recumbent posture. There may sometimes be definite salaam movements, and in a case which I saw at Westphal's clinic the patient incessantly made motions as if working a pump handle. After a period of intense bodily excitement, lasting for from twenty-four to thirty-six hours or longer, the patient can be examined, and presents the conditions which Bell described as typho-mania. The temperature ranges from 102° to 104°, or even higher. The tongue is dry, the pulse rapid and feeble, and sometimes there are seen on the skin bullæ and pustules, and frequently sores from abrasion and self-inflicted injuries. Toward the close or, according to Spitzka, even during the development of the disease there may be lucid intervals. There may be petechiæ on the skin, and often there is marked congestion of the face and extremities. The duration of the disease is variable. Very acute cases may terminate within a week; others persist for two or even three weeks. The course of the disease is almost uniformly fatal. The anatomical condition is practically negative, or at any rate presents nothing distinctive. There is great venous engorgement of the vessels of the meninges and of the gray cortex. In two cases in which I made a careful microscopic examination of the gray matter there were perivascular exudation and leucocytes in the lymph sheaths and perigangliar spaces. In the inspection of fatal cases of acute delirium careful examination should be made of the lungs and ileum. It should be borne in mind that in a majority of the cases dying in this manner, there is engorgement of the bases of the lungs or even deglutition pneumonia.

The nature of the disease is quite unknown. Some of the cases suggest acute infection. Spitzka thinks that it is due to an autochthonous nerve poison.

Diagnosis.—There are several diseases which may present identical symptoms. As Bell remarks in his paper, the first glance in many cases suggests typhoid fever, particularly when the patient is seen after the violence of the mania subsides. He gives two instances of this which were admitted from a general hospital. Enlargement of the spleen, the occurrence of spots, and the history give clews for the separation of the cases; but there are instances in which it is at first impossible to decide. More-

* American Journal of Insanity, 1849.

over, typhoid fever may set in with the most intense delirium. The existence of fever is the most deceptive symptom, and its combination with delirium and dry tongue so commonly means typhoid fever that it is very difficult to avoid error.

Acute pneumonia may come on with violent maniacal delirium and the pulmonary symptoms may be entirely masked.

Occasionally acute uræmia sets in suddenly with intense mania, and finally subsides into a fatal coma. The condition of the urine and the absence of fever would be important diagnostic features.

The character of the delirium is quite different from that of *mania à potu*. It may be extremely difficult to differentiate acute delirium from certain cases of cortical meningitis, which, however, is usually a secondary affection, occurring in connection with pneumonia or ulcerative endocarditis, or is due to extension from disease of the ear. This sets in more frequently with a chill, and there may be convulsions.

Treatment.—Even though bodily prostration is apt to come on early and be profound, I would not hesitate to advise, in the case of a robust man, free venesection. It is not at all improbable that some of the many cases of mania in which Benjamin Rush let blood with such benefit belonged to this class of affections. Considering its remarkable calming influence in febrile delirium, the cold bath or the cold pack should be employed. Morphia and chloroform may be administered, and hyoscine and the bromides may be tried. Krafft-Ebing states that Solivetti has obtained good results by the use of ergotin. Unfortunately, as asylum reports show, the disease is almost uniformly fatal.

II. PARALYSIS AGITANS

(*Parkinson's Disease; Shaking Palsy*).

Definition.—A chronic affection of the nervous system, characterized by muscular weakness, tremors, and rigidity.

Etiology.—Men are more frequently affected than women. It rarely occurs under forty, but instances have been reported in which the disease began about the twentieth year. It is by no means an uncommon affection. Direct heredity is rare, but the patients often belong to families in which there are other nervous affections. Among exciting causes may be mentioned exposure to cold and wet, and business worries and anxieties. In some instances the disease has followed directly upon severe mental shock or trauma. Cases have been described after the specific fevers. Malaria is believed by some to be an important factor, but of this there is no satisfactory evidence.

Morbid Anatomy.—No constant lesions have been found. The similarity between certain of the features of Parkinson's disease and those of old age suggest that the affection may depend upon a premature senil-

ity of certain regions of the brain. Our organs do not age uniformly, but in some, owing to hereditary disposition, the process may be more rapid than in others. "Parkinson's disease has no characteristic lesions, but on the other hand it is not a neurosis. It has for an anatomical basis the lesions of cerebro-spinal senility, and which only differ from those of true senility in their early onset and greater intensity." (Dubief.) The important changes are doubtless in the cerebral cortex.

Symptoms.—The disease begins gradually, usually in one or other hand, and the tremor may be either constant or intermittent. With this may be associated weakness or stiffness. At first these symptoms may be present only after exertion. Although the onset is slow and gradual in nearly all cases, there are instances in which it sets in abruptly after fright or trauma. When well established the disease is very characteristic, and the diagnosis can be made at a glance. The four prominent symptoms are tremor, weakness, rigidity, and the attitude.

Tremor.—This may be in the four extremities or confined to hands or feet; the head is not so commonly affected. The tremor is usually marked in the hands, and the thumb and forefinger display the motion made in the act of rolling a pill. At the wrist there are movements of pronation and supination, and less marked of flexion and extension. The upper-arm muscles are rarely involved. In the legs the movement is most evident at the ankle-joint, and less in the toes than in the fingers. Shaking of the head is less frequent, but does occur, and is usually vertical, not rotatory. The rate of oscillation is about five per second. Any emotion exaggerates the movement. The attempt at a voluntary movement may check the tremor (the patient may be able to thread a needle), but it returns with increased intensity. The tremors cease, as a rule, during sleep, but persist when the muscles are at repose. The writing of the patient is tremulous and zigzag.

Weakness.—Loss of power is present in all cases, and may occur even before the tremor, but is not very striking, as tested by the dynamometer, until the late stages. The weakness is greatest where the tremor is most developed. The movements, too, are remarkably slow. There is rarely complete loss of power.

Rigidity may early be expressed in a slowness and stiffness in the voluntary movements, which are performed with some effort and difficulty, and all the actions of the patient are deliberate. This rigidity is in all the muscles, and leads ultimately to the characteristic

Attitude and Gait.—The head is bent forward, the back is bowed, and the arms are held away from the body and are somewhat flexed at the elbow-joints. The face is expressionless, and the movements of the lips are slow. The eyebrows are elevated, and the whole expresion is immobile or mask-like, the so-called Parkinson's mask. The voice, as pointed out by Buzzard, is apt to be shrill and piping, and there is often a hesitancy in beginning a sentence; then the words are uttered with rapidity, as if the

patient was in a hurry. This is sometimes in striking contrast to the scanning speech of insular sclerosis. The fingers are flexed and in the position assumed when the hand is at rest; in the late stages they cannot be extended. Occasionally there is overextension of the terminal phalanges. The hand is usually turned toward the ulnar side, and the attitude somewhat resembles that of advanced cases of rheumatoid arthritis. In the late stages there are contractures at the elbows, knees, and ankles. The movements of the patient are characterized by great deliberation. He rises from the chair slowly in the stooping attitude, with the head projecting forward. In attempting to walk the steps are short and hurried, and, as Trousseau remarks, he appears to be running after his centre of gravity. This is termed festination or propulsion, in contradistinction to a peculiar gait observed when the patient is pulled backward, when he makes a number of steps and would fall over if not prevented—retropulsion.

The reflexes are normal in most cases, but in a few they are exaggerated.

Of sensory disturbances Charcot has noted abnormal alterations in the temperature sense. The patient may complain of subjective sensations of heat, either general or local—a phenomenon which may be present on one side only and associated with an actual increase of the surface temperature, as much as 6° F. (Gowers). In other instances, patients complain of cold. Localized sweating may be present. The mental condition rarely shows any change.

...ions in the Symptoms.—The tremor may be absent, but the rigi... ...eakness, and attitude are sufficient to make the diagnosis. The disease may be hemiplegic in character, involving only one side or even one limb. Usually these are but stages of the disease.

Diagnosis.—In well-developed cases the disease is recognized at a glance. The attitude, gait, stiffness, and mask-like expression are points of as much importance as the oscillations, and usually serve to separate the cases from senile and other forms of tremor. Disseminated sclerosis develops earlier, and is characterized by the nystagmus, and the scanning speech, and does not present the *attitude* so constant in paralysis agitans. The hemiplegic form might be confounded with post-hemiplegic tremor, but the history, the mode of onset, and the greatly increased reflexes would be sufficient to distinguish the two. The Parkinsonian face is of great importance in the diagnosis of the obscure and anomalous forms.

The disease is incurable. Periods of improvement may occur, but the tendency is for the affection to proceed progressively downward. It is a slow, degenerative process and the cases last for years.

Treatment.—There is no method which can be recommended as satisfactory in any respect. Arsenic, opium, and hyoscyamia may be tried, but the friends of the patient should be told frankly that the disease is incurable, and that nothing can be done except to attend to the physical comforts of the patient.

Other Forms of Tremor.

(*a*) *Simple Tremor.*—This is occasionally found in persons in whom it is impossible to assign any cause. It may be transient or persist for an indefinite time. It is often extremely slight, and is aggravated by all causes which lower the vitality.

(*b*) *Hereditary Tremor.*—C. L. Dana has reported remarkable cases of hereditary tremor. It occurred in all the members of one family, and beginning in infancy it continued without producing any serious changes.

(*c*) *Senile Tremor.*—With advancing age tremulousness during muscular movements is extremely common, but is rarely seen under seventy. It is always a fine tremor, which begins in the hands and often extends to the muscles of the neck, causing slight movement of the head.

(*d*) *Toxic tremor* is seen chiefly as an effect of tobacco, alcohol, lead, or mercury; more rarely in arsenical or opium poisoning. In elderly men who smoke much it may be entirely due to the tobacco. One of the commonest forms of this is the alcoholic tremor, which occurs only on movement and has considerable range. Lead tremor will be considered in speaking of lead poisoning, of which it constitutes a very important symptom.

(*e*) *Hysterical tremor*, which usually occurs under circumstances which make the diagnosis easy, will be considered in the section on hysteria.

III. ACUTE CHOREA

(*Sydenham's Chorea; St. Vitus's Dance*).

Definition.—A disease chiefly affecting children, characterized by irregular, involuntary contraction of the muscles, a variable amount of psychical disturbance, and a remarkable liability to acute endocarditis.

We shall speak here only of Sydenham's chorea. Senile chorea, chronic chorea, the prehemiplegic and post-hemiplegic forms, and rhythmic chorea are totally different affections.

Etiology.—*Sex.*—Of 554 cases which I have analyzed from the Philadelphia Infirmary for Diseases of the Nervous System, seventy-one per cent were in females and twenty-nine per cent in males. After puberty the percentage in females increases.

Age.—The age incidence in 522 cases was as follows: In the first decade, 201; in the second decade, 248; in the third decade, 10; in the fourth decade, 1; above the fourth decade, 2. In the cases under twenty years the following is the age incidence in the hemidecades: In the first hemidecade, 33; in the second hemidecade, 168; in the third hemidecade, 212; in the fourth hemidecade, 52.

Station.—While the disease affects children of all grades of society, it is more common among the lower classes.

Race.—As shown by inquiries instituted by Weir Mitchell some years ago, chorea is rare in the negro. No negro child of full birth has been under treatment at the Philadelphia Infirmary. From inquiries made among the medical men who practise in the Indian Territories and in the Indian schools in this country, I find that the disease is unknown in the native races

Seasonal Relations.—Morris J. Lewis has analyzed 437 separate attacks with reference to this point. Throughout December, January, and February the cases increase There is a fall in April, a rise through May and July, and then a steady fall until October. The cases are most numerous when the mean relative humidity and barometric pressure are low.

Rheumatism.—A causal relationship between rheumatism and chorea has been claimed by many since the time of Bright. The English and French writers maintain the closeness of this connection, and Roger goes so far as to regard the disease in all cases as a manifestation of rheumatism. On the other hand, German authors, as a rule, regard the connection as by no means very close. Discrepancy such as exists between the figures of Steiner, who found only 4 cases of acute rheumatism in 252 cases of chorea, and English writers, such as Dickenson, Barlow, and others, who place the percentage at from fifty to seventy of the cases, can only be explained on the supposition that the connection varies greatly in different localities. Of 554 cases which I have analyzed, in 15·5 per cent there was a history of rheumatism in the family. In 88 cases, 15·8 per cent, there was a history of articular swelling, acute or subacute. In 33 cases there were pains, sometimes described as rheumatic, in various parts, but not associated with joint trouble. If we regard all such cases as rheumatic and add them to those with manifest articular trouble, the percentage is raised to nearly twenty-one.

We find two groups of cases in which acute arthritis is present in chorea. In one, the arthritis antedates by some months or years the onset of the chorea, and does not recur before or during the attack. In the other group, the chorea sets in with or follows immediately upon the acute arthritis. In some instances it is impossible to decide whether the joint trouble or the movements come first. It is difficult to differentiate the cases of irregular pains without definite joint affection. It is probable that many of them are rheumatic, and yet I think it would be a mistake to regard as such all cases in children in which there are complaints of vague pains in the bones or muscles—so-called growing pains. It should never be forgotten, however, that a slight articular swelling may be the sole manifestation of rheumatism in a child—so slight indeed, that the disease may be entirely overlooked. The statistics of the Collective Investigation Committee of the British Medical Association, based upon 439 cases, give twenty-six per cent of antecedent joint affection, and if the cases of vague pains believed to be rheumatic are added, the percentage is raised to thirty-two. In this country rheumatism is not

so common in children as in England. Of the last 144 cases of the Infirmary series, almost every one of which I saw personally, and in which the most minute inquiries were made about rheumatism, there were only 25 cases with articular pains or swelling, and in only 6 had there been acute inflammatory rheumatism. The question may reasonably be asked, Do these articular affections of chorea belong to true rheumatism? Are they not analogous to the joint troubles of scarlet fever, puerperal fever, and gonorrhœa, which no one now regards as truly rheumatic? They have been spoken of by French writers as choreic arthropathies.

Heart-disease.—Endocarditis is believed by some writers to be the cause of the disease. The particles of fibrin and vegetations from the valves pass as emboli to the cerebral vessels. On this view, which we shall discuss later, chorea is the result of an embolic process occurring in the course of a rheumatic endocarditis.

Infectious Diseases.—Scarlet fever with arthritic manifestations may be a direct antecedent. It may be mentioned that a history of this disease occurred in 141 cases, or about twenty-five per cent. Sturges states that a history of previous whooping-cough occurs more frequently in choreic than in other children, but I find no evidence of this in the Infirmary records. With the exception of rheumatic fever, there is no intimate relationship between chorea and the acute diseases incident to childhood. It may be noted in contrast to this that the so-called canine chorea is a common sequel of distemper. Chorea has been known to develop in the course of an acute pyæmia, and to follow gonorrhœa and puerperal fever.

Kinnicutt and others have reported cases of chorea in malarial fevers, but the association was probably accidental, not causal. Anæmia is less often an antecedent than a sequence of chorea, and though cases develop in children who are anæmic and in poor health, this is by no means the rule. Chorea may develop in chlorotic girls at puberty.

Pregnancy.—Chorea may occur during pregnancy—most often during the first five months. It is more common in a first pregnancy, and is rare in women over twenty-five years of age. The disease is usually severe and maniacal symptoms may develop. Occasionally it comes on after an abortion or after delivery at term.

A tendency to the disease is found in certain families. In eighty cases there was a history of attacks of chorea in other members. In one instance both mother and grandmother had been affected. High-strung, excitable, nervous children are specially liable to the disease. *Fright* is considered a frequent cause, but in a large majority of the cases no close connection exists between the fright and the onset of the disease. Occasionally the attack sets in at once. Mental worry, trouble, a sudden grief, or a scolding may apparently be the exciting cause. The strain of *education*, particularly in girls during the third hemidecade, is a most important factor in the etiology of the disease. Bright, intelligent, active-minded girls from ten to fourteen, ambitious to do well at school, often stimulated

in their efforts by teachers and parents, form a large contingent of the cases of chorea in hospital and private practice. Sturges has called special attention to this *school-made* chorea as one serious evil in our modern method of forced education. *Imitation*, which is mentioned as an exciting cause, is extremely rare, and does not appear to have influenced the onset in a single case in the Infirmary records.

The disease may rapidly follow an injury or a slight surgical operation. Reflex irritation was believed to play an important *rôle* in the disease, particularly the presence of worms or genital irritation; but I have met with no instance in which the disease could be attributed to either of these causes. Local spasm, particularly of the face—the habit chorea of Mitchell—may be associated with irritation in the nostrils and adenoid growths in the vault of the pharynx, as pointed out by Jacobi.

It has been claimed by Stevens that *ocular defects* lie at the basis of many cases of chorea, and that with the correction of these the irregular movements disappear. To test the truth of these statements a careful study was made at the Infirmary by De Schweinitz of the condition of the eyes in 50 cases of chorea in children, with the following results: Hypermetropia was present in 23, or forty-six per cent; hypermetropia in one eye and hypermetropic astigmatism in the other in 7, or fourteen per cent; hypermetropic astigmatism in 12, or twenty-four per cent; myopia in 1, or two per cent; myopic astigmatism in 3, or six per cent; mixed astigmatism in 4, or eight per cent. De Schweinitz then adds the cases reported by Stevens and C. S. Bull, of New York, making a total of 227 cases, of which 112 were ametropic and 115 emmetropic. His conclusions are as follows: "Hypermetropia and hypermetropic astigmatism are vastly the preponderating condition in the eyes of choreic children, being found in about seventy-seven per cent of the cases, exactly as hypermetropic refraction is the preponderating condition in childhood, being found in seventy-six per cent of the eyes of children in the elementary schools," and the "evidence, however, seems quite as lacking that hypermetropic refraction is the basal cause of chorea, as it is that the chorea is the cause of the hypermetropia."

The committee of the New York Neurological Society which investigated with great care and impartiality Stevens's claims came to the conclusion that the facts did not warrant their adoption.

Morbid Anatomy and Pathology.—No constant lesions have been found in the nervous system in acute chorea. Vascular changes, such as hyaline transformation, exudation of leucocytes, minute hæmorrhages, and thrombosis of the smaller arteries, have been described.

Embolism of the smaller cerebral vessels has often been found, as might be expected in a disease with which endocarditis is so frequently associated. Based upon this fact, Kirkes, Tuckwell, Hughlings Jackson, and Bastian have supported what is known as the embolic theory of the disease. Endocarditis is by far the most frequent lesion in Sydenham's

chorea. With no disease, not excepting rheumatism, is it so constantly associated. In the records of over 110 autopsies, in nearly 100 this condition was mentioned. In the 5 autopsies of which I have notes, in all the mitral valves were affected. The endocarditis is usually of the simple variety, but the ulcerative form has occasionally been described.

We are still far from a solution of all the problems connected with chorea. Unfortunately, the word has been used to cover a series of totally diverse disorders of movement, so that there are still excellent observers who hold that chorea is only a symptom, and is not to be regarded as an etiological unit. The chorea of childhood, the disease which Sydenham described, presents, however, characteristics so unmistakable that it must be regarded as a definite, substantive affection. We cannot discuss fully, but only indicate briefly, certain of the theories which have been advanced with regard to it. The most generally accepted view is that it is a functional brain disorder affecting the nerve-centres controlling the motor apparatus, an instability of the nerve-cells, brought about, one supposes by hyperæmia, another by anæmia, a third by psychical influences, a fourth by irritation, centric or peripheric. Of the actual nature of this derangement we know nothing, nor, indeed, whether the changes are primary and the result of a faulty action of the cortical cells or whether the impulses are secondarily disturbed in their course down the motor path. The predominance of the disease in females, and its onset at a time when the education of the brain is rapidly developing, are etiological facts which Sturges has urged in favor of the view that chorea is an expression of functional instability of the nerve-centres.

The embolic theory originally advanced by Kirkes and supported by the English writers above mentioned has a solid basis of fact, but it is not comprehensive enough, as all of the cases cannot be brought within its limits. There are instances without endocarditis and without, so far as can be ascertained, plugging of cerebral vessels; and there are also cases with extensive endocarditis in which the histological examination of the brain, so far as embolism is concerned, was negative. In two of my post-mortems there were certainly no emboli in the smaller arteries of the branches of the circle of Willis or of the cortex. In the third there was a spot in one corpus striatum of red softening, probably due to an embolus. In favor of the embolic view is the experimental production in animals of chorea by Rosenthal, and later by Money, by injecting fine particles into the carotids of animals.

Lately, as indeed might be expected, a microbic origin has been sought for, and, however improbable such a theory looks at first sight, the case of tetanus gives a warrant, at least, to speculation and investigation in this direction. Nothing definite has yet been determined. From Naunyn's clinic a case is reported with endocarditis and a reddish-brown infiltration of the pia at the base of the brain which proved to be a microbic growth similar in character to those in the vegetations on the heart

valves. Recently, in a fatal case in my wards cultures of a micrococcus were obtained from the blood of the heart, and throughout the brain there were minute foci of hæmorrhage similar to those which occur in pneumonia and other infectious disease associated with endocarditis. In favor of this view it has been urged, as it is impossible to refer the chorea to endocarditis or the endocarditis in all cases to rheumatism, that both have their origin in a common cause, some infectious agent, which is capable also, in persons predisposed, of exciting articular disease. Cases have been reported in scarlet fever with arthritic manifestations, in puerperal fever, and rheumatism, also after gonorrhœa, and such facts are suggestive at least of the association of the disease with infective processes. Possibly, as has been suggested by some writers, the paralytic conditions associated with chorea may be analogous to those which occur in typhoid and certain of the infectious diseases. On the other hand, there are conditions extremely difficult to harmonize with this view. The prominent psychical element is certainly one of the most serious objections, since there can be no doubt that ordinary chorea may rapidly follow a fright or a sudden emotion. It cannot be supposed, too, that the forms associated with reflex irritation, as from the nose and particularly the cases of so-called habit chorea, can be dependent upon infection. We must place these in a separate category, and yet in a long series cases shade so imperceptibly into each other that it is extremely difficult to separate them properly. The question deserves careful study, and the possibility of a special infectious agent has of late been advocated by several writers.

Symptoms.—Three groups of cases may be recognized—the mild, severe, and maniacal chorea.

Mild Chorea.—In this the affection of the muscles is slight, the speech is not seriously disturbed, and the general health not impaired. Premonitory symptoms are shown in restlessness and inability to sit still, a condition well characterized by the term "fidgets." There are emotional disturbances, such as crying spells, or sometimes night-terrors. There may be pains in the limbs and headache. Digestive disturbances and anæmia may be present. A change in the temperament is frequently noticed, and a docile, quiet child may become cross and irritable. After these symptoms have persisted for a week or more the characteristic involuntary movements begin, and are often first noticed at the table, when the child spills a tumbler of water or upsets a plate. There may be only awkwardness or slight incoördination of voluntary movements, or constant irregular clonic spasms. The jerky, irregular character of the movements differentiates them from almost every other disorder of motion. In the mild cases only one hand, or the hand and face, are affected, and it may not spread to the other side.

In the second grade, the *severe form*, the movements become general and the patient may be unable to get about or to feed or undress herself, owing to the constant, irregular, clonic contractions of the various muscle

groups. The speech is also affected, and for days the child may not be able to talk. Often with the onset of the severer symptoms there is loss of power on one side or in the limb most affected.

The third and most extreme form, the so-called maniacal chorea, or *chorea insaniens*, is truly a terrible disease, and may develop out of the ordinary form. A young girl, aged eighteen, was admitted to the Montreal Hospital October 17. She was a waitress at a hotel, and being badly frightened by two men who were fighting, she dropped a tray of dishes which she was carrying. A severe reprimand increased her worry and trouble. The next day she packed her trunk and went home, a distance of thirty miles. Her father insisted that she should return. At this time her hands and arms began to twitch in a violent manner. Five days after the first fright she was admitted. The arms and legs were in constant motion, jerking in all directions. The face also was affected. She was rational, but could scarcely speak. On the night of the 19th she had no sleep, but raved and talked all the time, and the movements were incessant. On the 20th, 21st, and 22d the condition persisted and grew worse. The temperature ranged from 101° to 103°, the tongue became dry and cracked, and she became much exhausted. On the night of the 22d the temperature rose to 105° and death followed, ten days after the onset of the symptoms. These cases are more common in adult women and may develop during pregnancy.

Chorea begins, as a rule, in the hands and arms, then involves the face, and subsequently the legs. The movements may be confined to one side —hemichorea. The attack begins oftenest on the right side, though occasionally it is general from the outset. One arm and the opposite leg may be involved. In nearly one fourth of the cases speech is affected; when slight this is only an embarrassment or hesitancy, but in other instances it becomes an incoherent jumble. In very severe cases the child will make no attempt to speak. The inability is in articulation rather than in phonation. The lips and tongue are concerned in the defect. Occasionally the inspiratory muscles are involved, even when the speech is not at all affected, and sobbing and sighing may result. Paroxysms of panting and of hard expiration may occur, or odd sounds may be produced. As a rule the movements cease during sleep.

A prominent symptom is muscular weakness, usually no more than a condition of paresis. The loss of power is slight, but the weakness may be shown by an enfeebled grip or by a dragging of the leg or limping. In his original account Sydenham refers to the "unsteady movements of one of the legs, which the patient drags." There may be extreme paresis with but few movements—the paralytic chorea of Todd. Occasionally a local paralysis or weakness remains after the attack. Case 229 of the Infirmary series, a lad of ten, had severe general chorea in September, 1880, with considerable loss of power in the legs. Recovery was slow, and when he returned in September, 1883, in a second attack of chorea, there was talipes of the

left foot, which had resulted from paralysis in 1880. In Case 21 a wrist-drop persisted for two years, the result of a palsy which came on with chorea. These are probably instances of peripheral neuritis.

A question of some interest is whether choreic spasms extend to the muscles of organic life. The great gastro-intestinal muscle is never affected. There are no symptoms which can be referred to anomalous contractions of the stomach or bowels. The sphincters act normally. Incontinence of urine occurs occasionally, but it is not noted more frequently, I think, in chorea than in other nervous affections. Spasm of the bronchial muscles is not found even in severe cases, in which the respiratory muscles are involved. The pupils are usually dilated, but no irregular contractions occur. The rapid action and disturbed rhythm of the heart present nothing peculiar to the disease, and there is no support for the view that irregular contractions occur in the papillary muscles.

Heart Symptoms.—*Neurotic.*—As so many of the subjects of chorea are nervous girls, it is not surprising that a common symptom is rapidly acting heart. Any emotional disturbance causes at once a marked increase in the number of the beats, and the actions may become irregular and tumultuous. Irregularity, however, is not so special a feature in chorea as rapidity. The patients seldom complain of pain about the heart.

Hæmic Murmurs.—With anæmia and debility, not uncommon associates of chorea in the third and fourth week, we find a corresponding cardiac condition. The impulse is diffuse, perhaps wavy in thin children. The carotids throb visibly, and in the recumbent posture there may be pulsation in the cervical veins. On auscultation a systolic murmur is heard at the base, perhaps, too, at the apex, soft and blowing in quality.

Endocarditis.—As in rheumatism, so in chorea, acute valvulitis rarely gives evidence of its presence by symptoms. It must be sought, and clinical experience has shown that it is usually associated with murmurs at one or other of the cardiac orifices.

For the guidance of the practitioner the following statements may be made:

(1) In thin, nervous children a systolic murmur of soft quality is extremely common at the base, particularly at the second left costal cartilage, and is probably of no moment.

(2) A systolic murmur of maximum intensity at the apex, and heard also along the left sternal margin, is not uncommon in anæmic, enfeebled states, and does not necessarily indicate either endocarditis or insufficiency.

(3) A murmur of maximum intensity at apex, with rough quality, and transmitted to axilla or angle of scapula, indicates an organic lesion of the mitral valve, and is usually associated with signs of enlargement of the heart.

(4) When in doubt it is much safer to trust to the evidence of eye

and hand than to that of the ear. If the apex beat is in the normal position, and the area of dulness not increased vertically or to the right of the sternum, there is probably no serious valvular disease.

(5) The endocarditis of chorea is almost invariably of the simple or warty form, and in itself is not dangerous; but it is apt to lead to those sclerotic changes in the valve which produce incompetency. Of 110 choreic patients* examined more than two years after the attack, 54 presented signs of organic heart-disease.

(6) Pericarditis is an occasional complication of chorea, usually in cases with well-marked rheumatism.

Sensory Disturbances.—Pain in the affected limbs is not common. Occasionally there is soreness on pressure. There are cases, usually of hemichorea, in which pain in the limbs is a marked symptom. Weir Mitchell has spoken of these as *painful choreas*. The pain may be quite apart from any arthritic complications. Tingling and pricking sensations and numbness are found occasionally. Anæsthesia is very uncommon. Tender points along the lines of emergence of the spinal nerves or along the course of the nerves of the limbs are rare. The French writers have compared these to the hysterogenic points in hysteria, and have also described in certain cases ovarian tenderness. Headache may be a very troublesome symptom.

Psychical disturbances are common, though in a majority of the cases slight in degree. Irritability of temper, marked wilfulness, and emotional outbreaks may indicate a complete change in the character of the child. There is deficiency in the powers of concentration, the memory is enfeebled, and the aptitude for study is lost. Rarely there is progressive impairment of the intellect with termination in actual dementia. Acute melancholia has been described (Edes). Hallucinations of sight and hearing may occur. Patients may behave in an odd and strange manner and do all sorts of meaningless acts. By far the most serious manifestation of this character is the maniacal delirium, occasionally associated with the very severe cases—*chorea insaniens*. Usually the motor disturbance in these cases is aggravated, but it has been overlooked and patients have been sent to an asylum.

The psychical element in chorea is apt to be neglected by the practitioner. It is always a good plan to tell the parents that it is not the muscles alone of the child which are affected, but that the general irritability and change of disposition, so often found, really form part of the disease.

The condition of the *reflexes* in chorea is usually normal. Sinkler made observations at the Philadelphia Infirmary in 50 cases with the following results: In 26 the knee-jerk was normal, in 15 it was diminished in degree, and in 9 it could not be obtained. Trophic lesions rarely occur

* American Journal of the Medical Sciences, 1887, ii.

in chorea unless, as some writers have done, we regard the joint troubles as arthropathies occurring in the course of a cerebro-spinal disease.

Fever is not, as a rule, present in chorea unless complications exist. There may be the most intense and violent movements without any rise of temperature. I have seen instances, however, in which without apparently any visceral or articular disturbances there was slight daily fever. H. A. Hare states that in monochorea the temperature on the affected side may be elevated; but this is not an invariable rule. Fever is found with an acute arthritis, when there is marked endocarditis or pericarditis, though the former may certainly occur with little if any rise in temperature, and in the cases of maniacal chorea, in which the fever may range from 102° to 104°.

Cutaneous Affections.—These are not very numerous, and in a majority of the cases are probably due to arsenic. There may be an erythematous papular rash. A very interesting condition is the pigmentation which has been found in patients who have been taking arsenic for some time. Herpes zoster occasionally occurs. It was noted twice in the Infirmary records. Certain skin eruptions, usually regarded as rheumatic in character, are not uncommon. Thus, erythema nodosum has been described and I have seen several cases with a purpuric urticaria. There may, indeed, be the more aggravated condition of rheumatic purpura, known as Schönlein's *peliosis rheumatica.* Subcutaneous fibrous nodules, which have been noted by English observers in many cases of chorea, associated with rheumatism, are extremely rare in this country. I have not seen an instance in a choreic patient nor is there a reference in the Infirmary records to a case. This has not been because they were not looked for, as I have seen many instances since my attention was called to them in 1881 by Barlow at the Great Ormond Street Children's Hospital. They are certainly less common in this country than in England. In the chorea returns of the Collective Investigation Committee there were 12 cases out of 439. Cheadle states that they are not uncommon in chorea.

Duration and Termination.—From eight to ten weeks is the average duration of an attack of moderate severity. Cases may be so mild as to get well in two or three weeks; on the other hand, there may be found at every clinic for diseases of the nervous system choreic patients who have been under treatment for three, four, or even six months. Chronic chorea rarely follows the minor disease which we have been considering. The cases described under this designation in children are usually instances of cerebral sclerosis or Friedreich's ataxia; but occasionally an attack which has come on in the ordinary way persists for months or years, and recovery ultimately takes place. A slight grade of chorea, particularly noticeable under excitement, may persist for months in nervous children.

The tendency of chorea to recur has been noticed by all writers since Sydenham first made the observation. Of 410 cases analyzed for this pur-

pose, 240 had one attack, 110 had two attacks, 35 three attacks, 10 four attacks, 12 five attacks, and 3 six attacks. The recurrence is apt to be vernal. Rheumatism seems to favor this tendency; of 60 cases in which there were three or more attacks, there was a history of articular disease in 11, a much higher percentage than in cases with only one or two attacks. The occurrence of heart-disease has been thought to increase this liability, but I think it is the other way—recurrences tend to induce endocarditis and valvular disease. Gowers mentions a case with nine recurrences without history of rheumatism in which there were signs of mitral constriction.

Recovery is the rule in children. The statistics of out-patients' departments are not favorable for determining the mortality. A reliable estimate is that of the Collective Investigation Committee of the British Medical Association, in which 9 deaths were reported among 439 cases, about two per cent.

The paralysis rarely persists. Mental dulness may be present for a time, but usually passes away; permanent impairment of the mind is an exceptional sequence.

Diagnosis.—There are few diseases which present more characteristic features, and in a majority of instances the nature of the trouble is recognized at a glance; but there are several affections in children which may simulate and be mistaken for it.

(*a*) Multiple and diffuse cerebral sclerosis. Cases such as the following are often mistaken for ordinary chorea, and have been described in literature as *chorea spastica:* Nellie P., aged nine years, when two years old had fits which recurred constantly for twenty-one days and persisted on and off with great severity for nine months; she never developed satisfactorily; she learned to talk, but gradually began to have irregular movements. In the ninth year the condition was as follows: Speech hesitating; is unable to sit, stand, or feed herself; can move every muscle of the body, but in an irregular, incoördinate way, which prevents her from using any group of muscles. In attempting to grasp an object the fingers are thrown out in a stiff, spasmodic manner, and she is unable to close them over the object.

In such cases, which are not very uncommon, there are doubtless chronic changes in the cortex. As a rule, the movements are readily distinguishable from those of true chorea, but the simulation is sometimes very close; the onset in infancy, the impaired intelligence, increased reflexes, and in some instances rigidity and the chronic course of the disease, separate them sharply from true chorea.

(*b*) Friedreich's ataxia. Cases of this well-characterized disease were formerly classed as chorea. The slow, irregular, incoördinate movements, the scoliosis, scanning speech, the early talipes, the nystagmus, and the family character of the disease are points which should render the diagnosis easy.

(*c*) In rare cases the paralytic form of chorea may be mistaken for polio-myelitis or, when both legs are affected, for paraplegia of spinal origin; but this can only be the case when the choreic movements are very slight. I have at present under my care a young girl with chorea and loss of power in both legs, who was sent to the hospital as an instance of paraplegia due to spinal disease, but the choreic movements were distinct though slight, and a few days' observation sufficed to render clear the nature of the case.

(*d*) Hysteria may simulate chorea minor most closely, and unless there are other manifestations it may be impossible to make a diagnosis. Most commonly, however, the movements in the so-called hysterical chorea are rhythmic and differ entirely from those of ordinary chorea.

(*e*) As mentioned above, the mental symptoms in maniacal chorea may mask the true nature of the disease and patients have even been sent to the asylum.

Treatment.—Abnormally bright, active-minded children belonging to families with pronounced neurotic taint should be carefully watched from the ages of eight to fifteen and not allowed to overtax their mental powers. So frequently in children of this class does the attack of chorea date from the worry and stress incident to school examinations that the competition for prizes or places should be emphatically forbidden.

The treatment of the attack consists largely in attention to hygienic measures, with which alone, in time, a majority of the cases recover. Parents should be told to scan gently the faults and waywardness of choreic children. The psychical element, strongly developed in so many cases, is best treated by quiet and seclusion. The child should be confined to bed in the recumbent posture and mental as well as bodily quiet enjoined. In private practice this is often impossible, but with well-to-do patients the disease is always serious enough to demand the assistance of a skilled nurse. Toys and dolls should not be allowed at first, for the child should be kept amused without excitement. The rest allays the hyper-excitability and reduces to a minimum the possibility of damage to the valve segments should endocarditis exist. Time and again have I seen very severe cases which had resisted treatment for weeks outside a hospital become quiet and the movements subside after two or three days of absolute rest in bed.

The child should be kept apart from other children and, if possible, from other members of the family, and should see only those persons directly concerned with the nursing of the case. Though irksome and troublesome to carry out, this is an important part of the treatment. In the latter period of the disease daily rubbings may be resorted to with great benefit.

The medicinal treatment of the disease is unsatisfactory; with the exception of arsenic, no remedy seems to have any influence in controlling the progress of the affection. Without any specific action, it certainly does good in many cases, probably by improving the general

nutrition. It is conveniently given in the form of Fowler's solution, and the good effects are rarely seen until maximum doses are taken. Children stand the drug so well that I usually begin with five minims three times a day, and after three days increase the dose by one minim each day. When the dose of fifteen minims is reached, it may be continued for a week, and then again increased, if necessary, every day or two, until physiological effects are manifest. On the occurrence of these the drug should be stopped for three or four days. The practice of resuming the administration with smaller doses is rarely necessary, as tolerance is usually established and we can begin with the dose which the child was taking when the symptoms of saturation occurred. I have frequently given as much as twenty-five minims three times a day. Usually the signs of saturation are trivial but plain, and I have never seen any ill effects from the large doses, but I have heard recently of a case of arsenical neuritis due to the administration of Fowler's solution in chorea.

Of other medicines, strychnine, the zinc compounds, nitrate of silver, bromide of potassium, belladonna, chloral, and especially cimicifuga, have been recommended, and may be tried in obstinate cases.

For its tonic effect electricity is sometimes useful; but it is not necessary as a routine treatment. The question of gymnastics is an important one. Early in the disease, when the movements are active, it is not advisable; but during convalescence carefully graduated exercises are undoubtedly beneficial. It is not well, however, to send a choreic child to a school gymnasium, as the stimulus of the other children and the excitement of the romping, violent play is very prejudicial.

Other points in treatment may be mentioned. It is important to regulate the bowels and to attend carefully to the digestive functions. For the anæmia so often present preparations of iron are indicated.

In the severe cases with incessant movements, sleeplessness, dry tongue, and delirium, the important indication is to procure rest, for which purpose chloral may be freely given, and, if necessary, morphia. Chloroform inhalations may be necessary to subdue the intensity of the paroxysms, but the high rate of mortality in this class of cases illustrates how often our best endeavors are fruitless. The wet pack is sometimes very soothing and should be tried. As these patients are apt to sink rapidly into a low typhoid state with heart weakness, a supporting treatment is required from the outset.

Cases are found now and then which drag on from month to month without getting either better or worse and resist all modes of treatment. Change of air and scene is sometimes followed by rapid improvement, and in these cases the treatment by rest and seclusion should always be given a full trial.

In all cases care should be taken to examine the nostrils, and glaring ocular defects should be properly corrected either by glasses or, if necessary, by operation.

After the child has recovered from the attack, the parents should be warned that return of the disease is by no means infrequent, and is particularly liable to follow overwork at school or debilitating influences of any kind. These relapses are apt to occur in the spring. Sydenham advised purging in order to prevent the vernal recurrence of the disease.

IV. OTHER AFFECTIONS DESCRIBED AS CHOREA.

(*a*) **Chorea Major; Pandemic Chorea.**—The common name, St. Vitus's dance, applied to chorea has come to us from the middle ages, when under the influence of religious fervor there were epidemics characterized by great excitement, gesticulations, and dancing. For the relief of these symptoms, when excessive, pilgrimages were made, and, in the Rhenish provinces, particularly to the Chapel of St. Vitus in Zebern. Epidemics of this sort have occurred also during this century, and descriptions of them among the early settlers in Kentucky have been given by Robertson and Yandell. It was unfortunate that Sydenham applied the term chorea to an affection in children totally distinct from this chorea major, which and is in reality an hysterical manifestation under the influence of religious excitement.

(*b*) **Habit Spasm (Habit Chorea); Convulsive Tic** (of the French).

Two groups of cases may be recognized under the designation of habit spasm—one in which there is simply localized spasmodic movements, and the other in which, in addition to this, there are explosive utterances and psychical symptoms, a condition to which French writers have given the name *tic convulsif*.

(1) *Habit Spasm.*—This is found chiefly in childhood, most frequently in girls from seven to fourteen years of age (Mitchell). In its simplest form there is a sudden, quick contraction of certain of the facial muscles, such as rapid winking or drawing of the mouth to one side, or the neck muscles are involved and there are unilateral movements of the head. The head is given a sudden, quick shake, and at the same time the eyes wink. A not infrequent form is the shrugging of one shoulder. The grimace or movement is repeated at irregular intervals, and is much aggravated by emotion. A short inspiratory sniff is not an uncommon symptom. The cases are found most frequently in children who are "out of sorts," or who have been growing rapidly, or who have inherited a tendency to neurotic disorders. Allied to or associated with this are some of the curious tricks of children. A boy at my clinic was in the habit every few moments of putting the middle finger into the mouth, biting it, and at the same time pressing his nose with the forefinger. Hartley Coleridge is said to have had a somewhat similar trick, only he bit his arm. In all these cases the habits of the child should be examined carefully, the nose and vault of the pharynx thoroughly inspected, and the eyes accurately

tested. As a rule the condition is transient, and after persisting for a few months or longer gradually disappears. Occasionally a local spasm persists —twitching of the eyelids, or the facial grimace.

(2) *Tic Convulsif* (*Gilles de la Tourette's Disease*).—This remarkable affection, often mistaken for chorea, more frequently for habit spasm, is really a psychosis allied to hysteria, though in certain of its aspects it has the features of monomania. The disease begins, as a rule, in young children, occurring as early as the sixth year, though it may develop after puberty. There is usually a markedly neurotic family history. The special features of the complaint are:

(*a*) Involuntary muscular movements, usually affecting the facial or brachial muscles, but in aggravated cases all the muscles of the body may be involved and the movements may be extremely irregular and violent.

(*b*) Explosive utterances, which may resemble a bark or an inarticulate cry. A word heard may be mimicked at once and repeated over and over again, usually with the involuntary movements. To this the term *echolalia* has been applied. A much more distressing disturbance in these cases is *coprolalia*, or the use of bad language. A child of eight or ten may shock its mother and friends by constantly using the word *damn* when making the involuntary movements, or by uttering all sorts of obscene words. Occasionally actions are mimicked—*echokinesis*.

(*c*) Associated with some of these cases are curious mental disturbances; the patient becomes the subject of a form of obsession or a fixed idea. I was consulted recently about a young girl in whom the spasms were very slight, amounting only to twitching of the eyes and slight jerking of the shoulder, but who had a most pronounced grade of the fixed idea known as *arithmomania*. Almost every action, even the most trifling, was preceded by the counting of a certain number of figures. Before she went to bed she had to tap her heel upon the side of the bedstead a certain number of times; before drinking the tumbler had to be rotated eight or ten times, and then when set down again the same act was repeated. Before opening the door a certain number of knocks had to be given. The greatest difficulty was experienced in getting her to brush her hair, as it took her so long to count the necessary number of figures before she began. In other cases the fixed idea takes the form of the impulse to touch objects. According to Guinon, who has written an exhaustive article upon it in the Dictionnaire Encyclopédique, the prognosis is bad.

The disease is well marked and readily distinguished from ordinary chorea. The movements have a larger range and are explosive in character. Tourette regards the coprolalia as the most distinctive feature of the disease.

(*c*) **Saltatorie Spasm** (*Lata; Myriachit; Jumpers*).—Bamberger has described a disease in which when the patient attempted to stand there were strong contractions in the leg muscles, which caused a jumping or

springing motion. This occurs only when the patient attempts to stand. The affection has occurred in both men and women, more frequently in the former, and the subjects have usually shown marked neurotic tendencies. In many cases the condition has been transitory; in others it has persisted for years. Remarkable affections similar to this in certain points occur as a sort of endemic neurosis. One of the most striking of these occurs among the "jumping Frenchmen" of Maine and Canada. As described by Beard and Thornton, the subjects are liable on any sudden emotion to jump violently and utter a loud cry or sound, and will obey any command or imitate any action without regard to its nature. The condition of echolalia is present in a marked degree. The "jumping" prevails in certain families.

A very similar disease prevails in parts of Russia and in Java, where it is known by the names of myriachit and lata, the chief feature of which is mimicry by the patient of everything he sees or hears.

(*d*) **Chronic Chorea** (*Huntingdon's Chorea*).—An affection characterized by irregular movements, disturbance of speech, and gradual dementia. It is frequently hereditary. The disease has no connection with Sydenham's chorea, and it is unfortunate that the term was applied to it. It was described by Huntingdon, of Pomeroy, Ohio, at the time a practitioner on Long Island, and he gave in three brief paragraphs the salient points in connection with the disease—namely, the hereditary nature, the association with psychical troubles, and the late onset—between the thirtieth and fortieth years. The disease seems common in this country, and many cases have been reported by Clarence King, Sinkler, and others.* I have seen it in two Maryland families within the past two years. Under the term chronic chorea may be grouped the hereditary form and the cases which come on without family disposition, either at middle life or, more commonly, in the aged—senile chorea. It is doubtful whether the cases in children with chronic choreiform movements, often with mental weakness and spastic condition of the legs, should go into this category.

The hereditary character of the disease is very striking, and it has been traced through four or five generations. Huntingdon's father and grandfather, also physicians, had treated the disease in the family which he described. An identical affection occurs without any hereditary disposition. The age of onset is late, rarely before the thirtieth or the thirty-fifth year.

The symptoms are very characteristic. The irregular movements are usually first seen in the hands, and the patient has slight difficulty in performing delicate manipulations or in writing. When well established the movements are disorderly, irregular, incoördinate rather than choreic, and have not the sharp, brusque motion of Sydenham's chorea. In the face there are slow, involuntary grimaces. In a well-developed case the gait is irregular, swaying, and somewhat like that of a drunken man. The speech

* For complete literature, see Huet, de la Chorée Chronique, Paris, 1889.

is slow and difficult, the syllables are badly pronounced and indistinct, but not definitely staccato. The mental impairment is a gradual enfeeblement, leading finally to dementia. At first the patient may be emotional.

Very few post-mortems have been made. No characteristic lesions have been found. Atrophy of the convolutions, chronic meningo-encephalitis, and vascular changes have usually been present, the conditions which one would expect to find in a chronic dementia. These existed in an autopsy which I have on one of my cases. The affection is evidently a neuro-degenerative disorder, and has no connection with the simple chorea of childhood.

(*e*) **Rhythmic or Hysterical Chorea.**—This is readily recognized by the rhythmical character of the movements. It may affect the muscles of the abdomen, producing the salaam convulsion, or involve the sterno-mastoid, producing a rhythmical movement of the head, or the psoas, or any group of muscles. In its orderly rhythm it resembles the canine chorea.

V. INFANTILE CONVULSIONS (*Eclampsia*).

Convulsive seizures similar to those of epilepsy are not infrequent in children and in adults. The fit may indeed be identical with epilepsy, from which the condition differs in that when the cause is removed there is no tendency for the fits to recur. Occasionally, however, the convulsions in children continue and develop into true epilepsy.

Etiology.—A convulsion in a child may be due to many causes, all of which lead to an unstable condition of the nerve-centres, permitting of sudden, excessive and temporary nervous discharges. The following are the most important of them:

(1) Debility, resulting usually from gastro-intestinal disturbance. Convulsions frequently supervene toward the close of an attack of entero-colitis and recur, sometimes proving fatal. Morris J. Lewis has shown that the death rate in children from eclampsia rises steadily with that of gastro-intestinal disorders.

(2) Peripheral irritation. Dentition alone is rarely a cause of convulsions, but is often one of several factors in a feeble, unhealthy infant. The greatest mortality from convulsions is during the first six months, before the teeth really cut through the gums. Other irritative causes are the overloading of the stomach with indigestible food. It has been suggested that some of these cases are toxic, owing to the absorption of poisonous ptomaines. Worms, to which convulsions are so frequently attributed, probably have little influence. Among other sources possible are phimosis and otitis.

(3) Rickets. The observation of Sir William Jenner upon the association of rickets and convulsions has been amply confirmed. The spasms may be laryngeal, the so-called child-crowing, which, though convulsive in

nature, can scarcely be considered with eclampsia. The influence of this condition is more apparent in Europe than in this country, although rickets is a common disease, particularly among the colored people. Spasms, local or general, in rickets are probably associated with the condition of debility and malnutrition and with cranio-tabes.

(4) Fever. In young children the onset of the infectious diseases is frequently with convulsions, which often take the place of a chill in the adult. It is not known upon what they depend. Scarlet fever, measles, and pneumonia are most often preceded by convulsions.

(5) Congestion of the brain. That extreme engorgement of the blood-vessels may produce convulsions is shown by their occasional occurrence in severe whooping-cough, but their rarity in this disease really indicates how small a part mechanical congestion plays in the production of fits.

(6) Severe convulsions usher in or accompany many of the serious diseases of the nervous system in children. In more than fifty per cent of the cases of infantile hemiplegia the affection follows severe convulsions. They less frequently precede a spinal paralysis. They occur with meningitis, tuberculous or simple, and with tumors and other lesions of the brain.

And, lastly, convulsions may occur immediately after birth and persist for weeks or months. In such instances there has probably been meningeal hæmorrhage or serious injury to the cortex.

The most important question is the relation of convulsions in children to true epilepsy. In Gowers's figures of 1,450 cases of epilepsy, the attacks began in 180 during the first three years of life. Of 460 cases of epilepsy in children which I have analyzed, in 187 the fits began within the first three years. Of the total list the greatest number, 74, was in the first year. In nearly all these instances there was no interruption in the convulsions. That convulsions in early infancy are necessarily followed by epilepsy in after life is certainly a mistake.

Symptoms.—The attack may come on suddenly without any warning; more commonly it is preceded by a stage of restlessness, accompanied by twitching and perhaps grinding of the teeth. It is rarely so complete in its stages as true epilepsy. The spasm begins usually in the hands, most commonly in the right hand. The eyes are fixed and staring or are rolled up. The body becomes stiff and breathing is suspended for a moment or two by tonic spasm of the respiratory muscles, in consequence of which the face becomes congested. Clonic convulsions follow, the eyes are rolled about, the hands and arms twitch, or are flexed and extended in rhythmical movements, the face is contorted, and the head is retracted. The attack gradually subsides and the child sleeps or passes into a state of stupor. Following indigestion the attack may be single, but in rickets and intestinal disorders it is apt to be repeated. Sometimes the attacks follow each other with great rapidity, so that the child never rouses but dies

in a deep coma. If the convulsion has been limited chiefly to one side there may be slight paresis after recovery, or in instances in which the convulsions usher in infantile hemiplegia, when the child arouses one side is completely paralyzed. During the fit the temperature is often raised. Death rarely occurs from the convulsion itself, except in debilitated children or when the attacks recur with great frequency. In the so-called hydrocephaloid state in connection with protracted diarrhœa convulsions may close the scene.

Diagnosis.—Coming on when the subject is in full health, the attack is probably due either to overloaded stomach, to some peripheral irritation, or occasionally to trauma. Setting in with high fever and vomiting, it may indicate the onset of an exanthem, or occasionally be the primary symptom of encephalitis, or whatever the condition is which causes infantile hemiplegia. When the attack is associated with debility and with rickets the diagnosis is easily made. The carpopedal spasms and pseudo-paralytic rigidity which are often associated with rickets, laryngismus stridulus, and the hydrocephaloid state are usually confined to the hands and arms and are intermittent and usually tonic. The convulsions associated with tumor or which follow infantile hemiplegia are usually at first Jacksonian in character. After the second year convulsive seizures which come on irregularly without apparent cause and recur while the child is apparently in good health are likely to prove true epilepsy.

Prognosis.—Convulsions play an important part in infantile mortality. In Morris J. Lewis's table of deaths in children under ten, 8·5 per cent were ascribed to convulsions. West states that 22·35 per cent of deaths under one year are caused by convulsions, but this is too high an estimate for this country. In chronic diarrhœa convulsions are usually of ill omen. Those ushering in fevers are rarely serious, and the same may be said of the fits associated with indigestion and peripheral irritation.

Treatment.—Every source of irritation should be removed. If associated with indigestible food, a prompt emetic should be given, followed by an enema. The teeth should be examined, and if the gum is swollen, hot, and tense, it may be lanced; but never if it looks normal. When seen at first, if the paroxysm is severe, no time should be lost by giving a hot bath, but chloroform should be given at once, and repeated if necessary. A child is so readily put under chloroform and with such a small quantity that this precedure is quite harmless and saves much valuable time. The practice is almost universal of putting the child into a warm bath, and if there is fever the head may be douched with cold water. The temperature of the bath should not be above 95° or 96°. The very hot bath is not suitable, particularly if the fits are due to indigestion. After the attack an ice-cap may be placed upon the head. If there is much irritability, particularly in rickets and in severe diarrhœa, small doses of

opium will be found efficacious. When the convulsions recur after the child comes from under the influence of chloroform it is best to place it rapidly under the influence of opium, which may be given as morphia hypodemically, in doses of from one twenty-fifth to one thirtieth of a grain for a child of one year. Other remedies recommended are chloral by enema, in five-grain doses, and nitrite of amyl. After the attack has passed the bromides are useful, of which five to eight grains may be given in a day to a child a year old. Recurring convulsions, particularly if they come on without special cause, should receive the most thorough and careful treatment with bromides. When associated with rickets the treatment should be directed to improving the general condition.

VI. EPILEPSY.

Definition.—An affection of the nervous system characterized by attacks of unconsciousness, with or without convulsions.

The transient loss of consciousness without convulsive seizures is known as *petit mal;* the loss of consciousness with general convulsive seizures is known as *grand mal.* Localized convulsions, occurring usually without loss of consciousness, are known as epileptiform, or more frequently as Jacksonian or cortical epilepsy.

Etiology.—*Age.*—In a large proportion of all cases the disease begins before puberty. Of the 1,450 cases observed by Gowers, in 422 the disease began before the tenth year, and three fourths of the cases began before the twentieth year. Of 460 cases of epilepsy in children which I have analyzed * the age of onset in 427 was as follows: First year, 74; second year, 62; third year, 51; fourth year, 24; fifth year, 17; sixth year, 18; seventh year, 19; eighth year, 23; ninth year, 17; tenth year, 27; eleventh year, 17; twelfth year, 18; thirteenth year, 15; fourteenth year, 21; fifteenth year, 34. Arranged in hemidecades the figures are as follows: From the first to the fifth year, 229; from the fifth to the tenth year, 104; from the tenth to the fifteenth year, 95. These figures illustrate in a striking manner the early onset of the disease in a large proportion of the cases. It is well always to be suspicious of epilepsy developing in the adult, for in a majority of such cases the convulsions are due to a local lesion.

Sex.—No special influence appears to be discoverable in this relation, certainly not in children. Of 433 cases in my tables, 232 were males and 203 were females, showing a slight predominance of the male sex. After puberty unquestionably, if a large number of cases are taken, the males

* Three hundred and nine cases from the records from the Philadelphia Infirmary for Diseases of the Nervous System, 126 cases at the Elwyn Institution for Feeble-minded Children, and 25 from the records of my neurological clinic at the Johns Hopkins Hospital.

are in excess. The figures of Sieveking and Reynolds show that the disease is rather more prevalent in females than in males.

Heredity.—Much stress has been laid upon this by many authors as an important predisposing cause, and the statistics collected give from nine to over forty per cent. Gowers gives thirty-five per cent for his cases, which have special value apart from other statistics embracing large numbers of epileptics in that they were collected by him in his own practice. In our figures it appears to play a minor *rôle*. In the Infirmary list there were only 31 cases in which there was a history of marked neurotic taint, and only three in which the mother herself had been epileptic. In the Elwyn cases, as might be expected, the percentage is larger. Of the 126 there was in 32 a family history of nervous derangement of some sort, either paralysis, epilepsy, marked hysteria, or insanity. It is interesting to note that in this group, in which the question of heredity is carefully looked into, there were only two in which the mother had had epilepsy, and not one in which the father had been affected. Indeed, I was not a little surprised to find in the list of my cases that hereditary influences played so small a part. I have heard this opinion expressed by certain French physicians, notably Marie, who in writing also upon the question takes strong grounds against heredity as an important factor in epilepsy.

While, then, it may be said that direct inheritance is comparatively uncommon, the children of neurotic families in which neuralgia, insanity, and hysteria prevail are more liable to fall victim to the disease.

Chronic alcoholism in the parents is regarded by many as a potent predisposing factor in the production of epilepsy. Echeverria has analyzed 572 cases bearing upon this point and divided them into three classes, of which 257 cases could be traced directly to alcohol as a cause; 126 cases in which there were associated conditions, such as syphilis and traumatism; 189 cases in which the alcoholism was probably the result of the epilepsy. Figures equally strong are given by Martin,* who found in 150 insane epileptics 83 with a marked history of parental intemperance. Of the 126 Elwyn cases, in which the family history on this point was carefully investigated, in the majority of instances alcoholism seems not to have existed to any marked degree in the parents, a definite statement being found in only four of the cases.

Syphilis.—This in the parents is probably less a predisposing than an actual cause of epilepsy, which is the direct outcome of local cerebral manifestations. There is no reason for recognizing a special form of syphilitic epilepsy. On the other hand, convulsive seizures due to acquired syphilitic disease of the brain are very common.

Of exciting causes fright is believed to be important, but is less so, I think, than is usually stated. Trauma is present in a certain number of instances. An important group depends upon a local disease of the brain

* Annales Médicales Psychologiques, 1879.

existing from childhood, as seen in the post-hemiplegic epilepsy. Occasionally cases follow the infectious fevers. Masturbation has been stated to be a special cause, but its influence is probably overrated. A large group of convulsive seizures allied to epilepsy are due to some toxic agent, as in lead-poisoning and in uræmia. Great stress was laid upon reflex causes, such as dentition and worms, the irritation of a cicatrix, some local affection, such as adherent prepuce, or a foreign body in the ear or the nose. In many of these cases the fits cease after the removal of the cause, so that there can be no question of the association between the two. In others the attacks persist. Genuine cases of reflex epilepsy are, I believe, rare. A remarkable instance of it occurred at the Philadelphia Infirmary for Diseases of the Nervous System in the case of a man with a testis in the inguinal canal, pressure upon which would cause a typical fit. Removal of the organ was followed by cure.

Epilepsy has been thought to be associated with disturbance of the heart's action, and some have spoken of a special cardiac epilepsy, particularly in cases in which there is palpitation or slowing of the action prior to the onset. Epileptic seizures may occur during the passage of a gall-stone or occasionally during the removal of pleuritic fluid. Indigestion and gastric troubles are extremely common in epilepsy, and in many instances the eating of indigestible articles seems to precipitate an attack.

An attempt to associate genuine epilepsy with eye-strain has signally failed.

Symptoms.—(1) **Grand Mal.**—Preceding the fits there is usually a localized sensation, known as an *aura*, in some part of the body. This may be somatic, in which the feeling comes from some particular region in the periphery, as from the finger or hand, or is a sensation felt in the stomach or about the heart. The peripheral sensations preceding the fit are of great value, particularly those in which the aura always occurs in a definite region, as in one finger or toe. It is the equivalent of the signal symptom in a fit from a brain tumor. The varieties of these sensations are numerous. The epigastric sensations are most common. In these the patient complains of an uneasy sensation in the epigastrium or distress in the intestines, or the sensation may not be unlike that of heart-burn and may be associated with palpitation. These groups are sometimes known as pneumogastric auræ or warnings.

Of psychical auræ one of the most common, as described by Hughlings Jackson, is the vague, dreamy state, a sensation of strangeness or sometimes of terror. The auræ may be associated with special senses, of which the visual are the most common, consisting of flashes of light or sensations of color; less commonly, distinct objects are seen. The auditory auræ consist of noises in the ear, odd sounds, musical tunes, or occasionally voices. Olfactory and gustatory auræ, unpleasant tastes and odors, are rare.

Occasionally the fit may be preceded not by an aura, but by certain

movements; the patient may turn round rapidly or run with great speed for a few minutes, the so-called epilepsia procursiva. In one of the Elwyn cases the lad stood on his toes and twirled with extraordinary rapidity, so that his features were scarcely recognizable. At the onset of the attack the patient may give a loud scream or yell, the so-called epileptic cry. The patient drops as if shot, making no effort to guard the fall. In consequence of this, epileptics frequently injure themselves, cutting the face or head or burning themselves. In the attack, as described by Hippocrates, "the patient loses his speech and chokes, and foam issues from the mouth, the teeth are fixed, the hands are contracted, the eyes distorted, he becomes insensible, and in some cases the bowels are affected. And these symptoms occur sometimes on the left side, sometimes on the right, and sometimes on both." The fit may be described in three stages:

(*a*) *Tonic Spasm.*—The head is drawn back or to the right, and the jaws are fixed. The hands are clinched and the legs extended. This tonic contraction affects the muscles of the chest, so that respiration is impeded and the initial pallor of the face changes to a dusky or livid hue. The muscles of the two sides are unequally affected, so that the head and neck are rotated or the spine is twisted. The feet are extended and the knees and hip-joint are flexed. The arms are usually flexed at the elbows, the hand at the wrist, and the fingers are tightly clinched in the palm. This stage lasts only a few seconds, and then the

(*b*) *Clonic stage* begins. The muscular contractions become intermittent; at first tremulous or vibratory, they gradually become more rapid and the limbs are jerked and tossed about violently. The muscles of the face are in constant clonic spasm, the eyes roll, the eyelids are opened and closed convulsively. The movements of the muscles of the jaw are very forcible and strong, and it is at this time that the tongue is apt to be caught between the teeth and lacerated. The cyanosis, marked at the end of the tonic stage, gradually lessens. A frothy saliva, which may be blood-stained, escapes from the mouth. The fæces and urine may be discharged involuntarily. The duration of this stage is variable. It rarely lasts more than one or two minutes. The contractions become less violent and the patient gradually sinks into the condition of

(*c*) *Coma.* The breathing is noisy or even stertorous, the face congested, but no longer intensely cyanotic. The limbs are relaxed and the unconsciousness is profound. After a variable time the patient can be aroused, but if left alone he sleeps for some hours and then awakes, complaining only of slight headache or mental confusion.

In some cases one attack follows the other with great rapidity and consciousness is not regained. This is termed the *status epilepticus*, an exceptional condition, in which the patient may die of exhaustion consequent

upon the repeated attacks. In it the temperature is usually elevated. After the attack the reflexes are sometimes absent; more frequently they are increased and the ankle clonus can usually be obtained.

The state of the urine is variable, particularly as regards the solids. The quantity is usually increased after the attack, and albumen is not infrequently present.

Post-epileptic symptoms are of great importance. The patient may be in a trance-like condition, in which he performs actions of which subsequently he has no recollection. More serious are the attacks of mania, in which the patient is often dangerous and sometimes homicidal. It is held by good authorities that an outbreak of mania may be substituted for the fit. And, lastly, the mental condition of an epileptic patient is often seriously impaired, and profound defects are common.

Paralysis, which rarely follows the epileptic fit, is usually hemiplegic and transient.

Slight disturbances of speech also may occur; in some instances forms of sensory aphasia.

The attacks may occur at night, and a person may be epileptic for years without knowing it. As Trousseau truly remarks, when a person tells us that in the night he has incontinence of urine and awakes in the morning with headache and mental confusion, and complains of difficulty in speech owing to the fact that he has bitten his tongue; if, also, there are on the skin of the face and neck purpuric spots, the probability is very strong indeed that he is subject to nocturnal epilepsy.

(2) **Petit Mal.**—This is epilepsy without the convulsions. The attack consists of transient unconsciousness, which may come on at any time, accompanied or unaccompanied by a feeling of faintness and vertigo. Suddenly, for example, at the dinner table, the subject stops talking and eating, the eyes become fixed, and the face slightly pale. Anything which may have been in the hand is usually dropped. In a moment or two consciousness is regained and the patient resumes conversation as if nothing had happened. In other instances there is slight incoherency or the patient performs some almost automatic action. He may begin to undress himself and on returning to consciousness find that he has partially disrobed. In other attacks the patient may fall without convulsive seizures. A definite aura is rare. Though transient, unconsciousness and giddiness are the most constant manifestations of *petit mal;* there are many other equivalent manifestations, such as sudden jerkings in the limbs, sudden tremor, or a sudden visual sensation. Gowers mentions no less than seventeen different manifestations of *petit mal.*

After the attack the patient may be dazed for a few seconds and perform certain automatic actions, which may seem to be volitional. As mentioned, undressing is a common action, but all sorts of odd actions may be performed, some of which are awkward or even serious. One of my patients after an attack was in the habit of tearing anything he could lay

hands on, particularly books. Violent actions have been committed and assaults made, frequently giving rise to questions which come before the courts. This condition has been termed masked epilepsy, or *epilepsia larvata.*

In a majority of the cases of *petit mal* convulsions finally occur, at first slight, but ultimately the *grand mal* becomes well developed, and the attacks may then alternate.

(3) **Jacksonian Epilepsy.**—This is also known as cortical, symptomatic, or partial epilepsy. It is distinguished from the ordinary epilepsy by the important fact that consciousness is retained. The attacks are usually the result of irritative lesions in the motor zone, though there are probably also sensory equivalents of this motor form. In a typical attack the spasm begins in a limited muscle group of the face, arm, or leg. The zygomatic muscles, for instance, or the thumb may twitch, or the toes may first be moved. Prior to the twitching the patient may feel a sensation of numbness or tingling in the part affected. The spasm extends and may involve the muscles of one limb only or of the face. The patient is conscious throughout and watches, often with interest, the march of the spasm.

The onset may be slow, and there may be time, as in a case which I have reported, for the patient to place a pillow on the floor, so as to be as comfortable as possible during the attack. The spasms may be localized for years, but there is a great risk that the partial epilepsy may become general. The condition is due, as a rule, to an irritative lesion in the motor zone. Thus of 107 cases analyzed by Roland, there were 48 of tumor, 21 instances of inflammatory softening, 14 instances of acute and chronic meningitis, and 8 cases of trauma. The remaining instances were due to hæmorrhage or abscess, or were associated with sclerosis cerebri. Two other conditions may be mentioned, which may cause typical Jacksonian epilepsy—namely, uræmia and progressive paralysis of the insane. A considerable number of the cases of Jacksonian epilepsy are found in children following hemiplegia, the so-called post-hemiplegic epilepsy. The convulsions usually begin on the affected side, either in the arm or leg, and the fit may be unilateral and without loss of consciousness. Ultimately they become more severe and general.

Diagnosis.—In major epilepsy the suddenness of the attack, the abrupt loss of consciousness, the order of the tonic and clonic spasm, and the relaxation of the sphincters at the height of the attack are distinctive features. The convulsive seizures due to uræmia are epileptic in character and usually readily recognized by the existence of greatly increased tension and the condition of the urine. Practically in young adults hysteria causes the greatest difficulty, and may closely simulate true epilepsy. The following table from Gowers's work draws clearly the chief differences between them:

	Epileptic.	Hysteroid.
Apparent cause.....	none.	emotion.
Warning.....	any, but especially unilateral or epigastric auræ.	palpitation, malaise, choking, bilateral foot aura.
Onset.....	always sudden.	often gradual.
Scream.....	at onset.	during course.
Convulsion	rigidity followed by "jerking," rarely rigidity alone.	rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting.....	tongue.	lips, hands, or other people and things.
Micturition.....	frequent.	never.
Defecation.....	occasional.	never.
Talking.....	never.	frequent.
Duration.....	a few minutes.	more than ten minutes, often much longer.
Restraint necessary..	to prevent accident.	to control violence.
Termination.....	spontaneous.	spontaneous or induced (water, etc.).

Recurring epileptic seizures in a person over thirty who has not had previous attacks is always suggestive of organic disease. According to H. C. Wood, whose opinion is supported by that of Fournier, in nine cases out of ten the condition is due to syphilis.

Petit mal must be distinguished from attacks of syncope, and the vertigo of Menière's disease, of a cardiac lesion, and of indigestion. In these cases there is no actual loss of consciousness, which forms a characteristic though not an invariable feature of *petit mal.*

Jacksonian epilepsy has features so distinctive and peculiar that it is at once recognized. It is by no means easy, however, always to determine upon what the spasm depends. Irritation in the motor centres may be due to a great variety of causes, among which tumors and localized meningo-encephalitis are the most frequent; but it must not be forgotten that in uræmia localized epilepsy may occur. The most typical Jacksonian spasms also are not infrequent in general paresis of the insane.

Prognosis.—This may be given to-day in the words of Hippocrates: "The prognosis in epilepsy is unfavorable when the disease is congenital, and when it endures to manhood, and when it occurs in a grown person without any previous cause. . . . The cure may be attempted in young persons, but not in old."

Death during the fit rarely occurs, but it may happen if the patient falls into the water or if the fit comes on while he is eating. Occasionally the fits seem to stop spontaneously. This is particularly the case in the epilepsy in children which has followed the convulsions of teething or of the fevers. Frequency of the attacks and marked mental disturbance are unfavorable indications. Hereditary predisposition is apparently of no moment in the prognosis. The outlook is better in males than in females. The post-hemiplegic epilepsy is rarely arrested. Of the cases coming on

in adults, those due to syphilis and to local affections of the brain allow a more favorable prognosis.

Treatment.—*General.*—In the case of children the parents should be made to understand from the outset that epilepsy in the great majority of cases is an incurable affection, so that the disease may interfere as little as possible with the education of the child. The subjects need firm but kind treatment. Indulgence and yielding to caprices and whims are followed by weakening of the moral control, which is so necessary in these cases. The disease does not incapacitate a person for all occupation. It is much better for epileptics to have some definite pursuit. There are many instances in which they have been persons of extraordinary mental and bodily vigor; as, for example, Julius Cæsar and Napoleon. One of the most distressing features in epilepsy is the gradual mental impairment which follows in a certain number of cases. If such patients become extremely irritable or show signs of violence they should be placed under supervision in an asylum. Marriage should be forbidden to epileptics. During the attack a cork or bit of rubber should be placed between the teeth and the clothes should be loosened. The patient should be in the recumbent posture. As the attack usually passes off with rapidity, no special treatment is necessary, but in cases in which the convulsion is prolonged a few whiffs of chloroform or nitrite of amyl or a hypodermic of a quarter of a grain of morphia may be given.

Dietetic.—The old authors laid great stress upon regimen in epilepsy. The important point is to give the patient a light diet at fixed hours, and on no account to permit overloading of the stomach. Meat should not be given more than once a day. There are cases in which animal food seems injurious. A strictly vegetable diet has been warmly recommended. The patient should not go to sleep until the completion of gastric digestion.

Medicinal.—The bromides are the only remedies which have a special influence upon the disease. Either the sodium or potassium salt may be given. Sodium bromide is probably less irritating and is better borne for a long period. It may be given in milk, in which it is scarcely tasted. In all instances the dilution should be considerable. In adults it is well taken in soda water or in some mineral water. The dose for an adult should be from half a drachm to a drachm and a half daily. As Seguin recommends, it is often best to give but a single dose daily, about four to six hours before the attacks are most likely to occur. For instance, in the case of nocturnal epilepsy a drachm should be given an hour or two after the evening meal. If the attack occurs early in the morning, the patient should take a full dose when he awakes. When given three times a day it is best given after meals. Each case should be carefully studied to determine how much bromide should be used. The individual susceptibility varies and some patients require more than others. Fortunately, children take the drug well and stand proportionately larger doses than

adults. Saturation is indicated by certain unpleasant effects, particularly drowsiness, mental torpor, and gastric and cardiac distress. Loss of palate reflex is one of the earliest indications that the system is under the influence of the bromides, and is a condition which should be attained. A very unpleasant feature is the development of acne, which, however, is no indication of bromism. Seguin states that the tendency to this is much diminished by giving the drug largely diluted in alkaline waters and administering from time to time full doses of arsenic. To be effectual the treatment should be continued for a prolonged period and the cases should be incessantly watched in order to prevent bromism. The medicine should be continued for at least two years after the cessation of the fits; indeed, Seguin recommends that the reduction of the bromides should not be begun until the patient has been three years without any manifestations. Written directions should be given to the mother or to the friends of the patient, and he should not himself be held responsible for the administration of the medicine. A book should be provided in which the daily number of attacks and the amount of medicine taken should be noted.

Among other remedies which have been recommended as controlling epilepsy are chloral, cannabis indica, zinc, nitroglycerin, and borax. Nitroglycerin is sometimes advantageous in *petit mal*, but is not of much service in the major form. To be beneficial it must be given in full doses, from two to five minims of the one per cent solution, and increased until the physiological effects are produced. Counter-irritation is rarely advisable. When the aura is very definite and constant in its onset, as from the hand or from the toe, a blister about the part or a ligature tightly applied may stop the oncoming fit. In children, care should be taken that there is no source of peripheral irritation. In boys, adherent prepuce may occasionally be the cause. The irritation of teething, the presence of worms, and foreign bodies in the ears or nose have been associated with epileptic seizures.

The subjects of a chronic and, in most cases, a hopelessly incurable disease, epileptic patients form no small portion of the unfortunate victims of charlatans and quacks, who prescribe to-day, as in the time of the father of medicine, "purifications and spells and other illiberal practices of like kind."

Surgical.—In Jacksonian epilepsy the propriety of surgical interference is universally granted. It is questionable, however, whether in the epilepsy following hemiplegia, considering the anatomical condition, it is likely to be of any benefit. In idiopathic epilepsy, when the fit starts in a certain region—the thumb, for instance—and the signal symptom is invariable, the centre controlling this part may be removed. This procedure has been practised by Macewen, Horsley, Keen, and others, but time alone can determine its value. The traumatic epilepsy, in which the fit follows fracture, is much more hopeful.

The operation, *per se*, appears in some cases to have a curative effect. Thus of 50 cases of trephining for epilepsy in which nothing abnormal was found to account for the symptoms, 25 were reported as cured and 18 as improved.* The operations have not been always on the skull, and White has collected an interesting series in which various surgical procedures have been resorted to, often with curative effect, such as ligation of the carotid artery, castration, tracheotomy, excision of the superior cervical ganglia, incision of the scalp, circumcision, etc.

VII. MIGRAINE (*Hemicrania; Sick Headache*).

Definition.—A paroxysmal affection characterized by severe headache, usually unilateral, and often associated with disorders of vision.

Etiology.—The disease is frequently hereditary and has occurred through several generations. Women and the members of neurotic families are most frequently attacked. It is an affection from which many distinguished men have suffered and have left on record an account of the disease, notably the astronomer Airy. Edward Liveing's work is the standard authority upon which most of the subsequent articles have been based. A gouty or rheumatic taint is present in many instances. Sinkler has called special attention to the frequency of reflex causes. Migraine has long been known to be associated with uterine and menstrual disorders. Many of the headaches from eye-strain are of the hemicranial type. Brunton refers to caries of the teeth as a cause of these headaches, even when not associated with toothache. Cases have been described in connection with adenoid growths in the pharynx, and particularly with abnormal conditions of the nose. Many of the attacks of severe headaches in children are of this nature, and the eyes and nostrils should be examined with great care. Sinkler refers to a case in a child of two years, and Gowers states that a third of all the cases begin between the fifth and tenth years of age. The direct influences inducing the attack are very varied. Powerful emotions of all sorts are the most potent. Mental or bodily fatigue, digestive disturbances, or the eating of some particular article of food may be followed by the headache. The paroxysmal character is one of the most striking features, and the attacks may recur on the same day every week, every fortnight, or every month.

Symptoms.—Premonitory signs are present in many cases, and the patient can tell when an attack is coming on. Remarkable prodromata have been described, particularly in connection with vision. Apparitions may appear—visions of animals, such as mice, dogs, etc. Transient hemianopia or scotoma may be present. In other instances there is spasmodic action of the pupil on the affected side, which dilates and contracts

* J. William White, Curative Effects of Operations *per se*, Annals of Surgery, 1891.

alternately, the condition known as *hippus*. Frequently the disturbance of vision is only a blurring, or there are balls of light, or zigzag lines, or the so-called fortification spectra (teichopsia), which may be illuminated with gorgeous colors. Disturbances of the other senses are rare. Numbness of the tongue and face and occasionally of the hand may occur with tingling. More rarely there are cramps or spasms in the muscles of the affected side. Transient aphasia has also been noted. Some patients show marked psychical disturbance, either excitement or, more commonly, mental confusion or great depression. Dizziness occurs in some cases. The headache follows a short time after the prodromal symptoms have appeared. It is cumulative and expansile in character, beginning as a localized small spot, which is generally constant either on the temple or forehead or in the eyeball. It is usually described as of a penetrating, sharp, boring character. At first unilateral, it gradually spreads and involves the side of the head, sometimes the neck, and the pains may pass into the arm. In other cases both sides are affected. Nausea and vomiting are common symptoms. If the attack comes on when the stomach is full, vomiting usually gives relief. Vaso-motor symptoms may be present. The face, for instance, may be pale, and there may be a marked difference between the two sides. Subsequently the face and ear on the affected side may become a burning red from the vaso-dilator influences. The pulse may be slow. The temporal artery on the affected side may be firm and hard, and in a condition of arterio-sclerosis—a fact which has been confirmed anatomically by Thoma. Few affections are more prostrating than migraine, and during the paroxysm the patient may scarcely be able to raise the head from the pillow. The slightest noise or light aggravates the condition.

The duration of the entire attack is variable. The severer forms usually incapacitate the person for at least three days. In other instances the entire attack is over in a day. The disease recurs for years, and in cases with a marked hereditary tendency may persist throughout life. In women the attacks often cease after the climacteric, and in men after the age of fifty. Two of the greatest sufferers I have known, who had recurring attacks every few weeks from early boyhood, now have complete freedom.

The nature of the disease is unknown. Liveing's view, that it is a nerve storm or form of periodic discharge from certain sensory centres and is related to epilepsy, has found much favor. According to this view, it is the sensory equivalent of a true epileptic attack. Mollendorf, Latham, and others regard it as a vaso-motor neurosis, and hold that the early symptoms are due to vaso-constrictor and the later symptoms to vaso-dilator influences. The fact of the development of arterio-sclerosis in the arteries of the affected side is a point of interest bearing upon this view.

Treatment.—The patient is fully aware of the causes which precipitate an attack. Avoidance of excitement, regularity in the meals, and moderation in diet are important rules. The treatment should be directed

toward the removal of the conditions upon which the attacks depend. In children much may be done by watchfulness and care on the part of the mother in regulating the bowels and watching the diet of the child. Errors of refraction should be adjusted. On no account should such children be allowed to compete in school for prizes. A prolonged course of bromides sometimes proves successful. If anæmia is present, iron and arsenic should be given. When the arterial tension is increased a course of nitroglycerin may be tried. Not too much, however, should be expected of the preventive treatment of migraine. It must be confessed that in a very large proportion of the cases the headaches recur in spite of all we can do. During the paroxysm the patient should be kept in bed and absolutely quiet. If the patient feels faint and nauseated, a small cup of hot, strong coffee or twenty drops of chloroform give relief. Cannabis indica is probably the most satisfactory remedy. Seguin recommends a prolonged course of the drug. Antipyrin, antifebrin, and phenacetin have been much used of late. When given early, at the very outset of the paroxysm, they are sometimes effective. The doses which have been recommended of antifebrin and antipyrin are often dangerous, and I have seen in a case of migraine unpleasant collapse symptoms follow a twenty-five-grain dose of antipyrin which the patient had taken on her own responsibility. Smaller, repeated doses are more satisfactory. Of other remedies, caffeine, in five-grain doses of the citrate, nux vomica, and ergot have been recommended. Electricity does not appear to be of much service.

VIII. NEURALGIA.

Definition.—A painful affection of the nerves, due either to functional disturbance of their central or peripheral extremities or to neuritis in their course.

Etiology.—Members of neuropathic families are most subject to the disease. It affects women more than men. Children are rarely attacked. Of all causes, debility is the most frequent. It is often the first indication of an enfeebled nervous system. The various forms of anæmia are frequently associated with neuralgia. It may be a prominent feature at the onset of certain acute diseases, particularly typhoid fever. Malaria is believed to be a potent cause, but it has not been shown that neuralgia is more frequent in malarial districts, and the error has probably arisen from regarding periodicity as a special manifestation of paludism. It occasionally occurs in malarial cachexia. Exposure to cold is a cause in very susceptible persons. Reflex irritation, particularly from carious teeth, may induce neuralgia of the fifth nerve. The disease occurs sometimes in rheumatism, gout, lead poisoning, and diabetes.

Symptoms.—Before the onset of the pain there may be uneasy sensations, sometimes tingling in the part which will be affected. The pain

is localized to a certain group or division of nerves, usually affecting one side. The pain is not constant, but paroxysmal, and is described as stabbing, burning, or darting in character. The skin may be exquisitely tender in the affected region, particularly in certain points along the course of the nerve, the so-called tender points. Movements, as a rule, are painful. Trophic and vaso-motor changes may accompany the paroxysm; the skin may be cool, and subsequently hot and burning, occasionally local œdema or erythema occurs. More remarkable still are the changes in the hair, which may become blanched (canities), or even fall out. Fortunately, such alterations are rare. Twitchings of the muscles, or even spasms, may be present during the paroxysm. After lasting a variable time—from a few minutes to many hours—the attack subsides. Recurrence may be at definite intervals—every day at the same hour, or at intervals of two, three, or even seven days. Occasionally the paroxysms develop only at the catamenia. This periodicity is quite as marked in non-malarial as in malarial regions.

Clinical Varieties, depending on the Nerve Groups affected.—(1) *Trifacial Neuralgia; Tic Douloureux; Prosopalgia.*—All the branches are rarely involved together. The ophthalmic is most often affected, but in severe attacks the pains, though more intense in one division, radiate over the other branches. At the outset there may be hyperæsthesia of the skin and sensitiveness of the mucous membrane. Pressure is painful at the points of emergence of the nerve trunk, and where the nerves enter the muscles. Sometimes in addition, as Trousseau pointed out, there are pains at the occipital protuberance and in the upper cervical spines. When the ophthalmic division is affected the eye may weep and the conjunctivæ are injected and painful. In the upper maxillary division there is a tender point where the nerve leaves the infraorbital canal, and the pain is specially marked along the upper teeth. In the lower branches, which are more frequently involved, there are painful points along the auriculo-temporal nerve and the pain radiates in the region of the ear along the lower jaw and teeth. The movements of mastication and speaking may be painful. Salivation is not uncommon. Herpes may occur about the eye or the lips. In protracted cases there may be atrophy or induration of the skin. Some of the forms of facial neuralgia are of frightful intensity and the recurring attacks render the patient's life almost insupportable.

(2) *Cervico-occipital neuralgia* involves the posterior branches of the first four cervical nerves, particularly the inferior occipital, at the emergence of which there is a painful point about half-way between the mastoid process and the first cervical vertebra. It may be caused by cold, and these nerves are often affected in cervical caries.

(3) *Cervico-brachial neuralgia* involves the sensory nerves of the brachial plexus, particularly in the cubital division. When the circumflex nerve is involved the pain is in the deltoid. The pain is most commonly

about the shoulder and down the course of the ulnar nerve. There is usually a marked tender point upon this nerve at the elbow. This form rarely follows cold, but more frequently results from rheumatic affections of the joints, and trauma.

(4) *Neuralgia of the phrenic nerve* is rare. It is sometimes found in pleurisy and in pericarditis. The pain is chiefly at the lower part of the thorax on a line with the insertion of the diaphragm, and here may be painful points on deep pressure. Full inspiration is painful, and there is great sensitiveness on coughing or in the performance of any movement by which the diaphragm is suddenly depressed.

(5) *Intercostal Neuralgia.*—Next to the *tic douloureux* this is the most important form. It is most frequent in women and very common in hysteria and anæmia. The pain in caries and aneurism is felt in the intercostal nerves. They are also the seat of the intense pain in inflammation of the pleura. The pain is often constant and exaggerated by movements. Pleurodynia is supposed by some to be local intercostal neuralgia, confined to one spot, usually along the course or at the exit of the nerves. Herpes zoster or zona occurs with the most aggravated form of intercostal neuralgia. The pain usually precedes the eruption, which consists of a series of pearly vesicles, which take two or three days to develop and gradually disappear. The eruption may occur without much pain. The most distressing feature in the complaint is the persistence in the pain after the eruption has subsided. The eruption and the neuralgia are in reality manifestations of neuritis. Changes have been found in the nerves and in the ganglia of the posterior roots. The pain of zona may persist indefinitely, and it has been known to be so intractable that in despair the person has committed suicide.

(6) *Lumbar Neuralgia.*—The affected nerves are the posterior fibres of the lumbar plexus, particularly the ilio-scrotal branch. The pain is in the region of the iliac crest, along the inguinal canal, in the spermatic cord, and in the scrotum or labium majus. The affection known as irritable testis, probably a neuralgia of this nerve, may be very severe and accompanied by syncopal sensations.

(7) *Coccydynia.*—This is regarded as a neuralgia of the coccygeal plexus. It is most common in women, and is aggravated by the sitting posture. It is very intractable, and may necessitate the removal of the coccyx, an operation, however, which is not always successful. Neuralgias of the nerves of the leg have already been considered.

(8) *Neuralgias of the Nerves of the Feet.*

Painful Heel.—Both in women and men there may be about the heel severe pains which interfere seriously with walking—the pododynia of S. D. Gross. There may be little or no swelling, no discoloration, and no affection of the joints. The pain is usually most severe over the heel; sometimes in a very limited spot, sometimes in the line of the metatarso-phalangeal joint. Probably this painful affection depends upon many

different conditions. It may be associated with rheumatism or gout, and with certain occupations—persons who have to stand for a long time on their feet. In other instances it occurs with flat-foot.

Plantar Neuralgia.—This is often associated with a definite neuritis, such as follows typhoid fever, and has been seen in an aggravated form in caisson disease (Hughes). The pain may be limited to the tips of the toes or to the ball of the great toe. Numbness, tingling, and hyperæsthesia or sweating may occur with it. Following the cold-bath treatment in typhoid fever it is not uncommon for patients to complain of great sensitiveness in the toes.

Erythromelalgia.—Under this term Weir Mitchell described a condition which is associated with great pain in the heel or in the sole of the foot, with vascular changes, either an acute hyperæmia or cyanosis. Some of the cases should unquestionably be regarded as Raynaud's disease.

(9) *Visceral Neuralgias.*—The more important of these have already been referred to in connection with the cardiac and the gastric neuroses. They are most frequent in women, and are constant accompaniments of neurasthenia and hysteria. The pains are most common in the pelvic region, particularly about the ovaries. Nephralgia is of great interest, for, as has already been mentioned, the symptoms may closely simulate those of stone.

Treatment.—Causes of reflex irritation should be carefully removed. The neuralgia, as a rule, recurs unless the general health improves; so that tonic and hygienic measures of all sorts should be employed. Often a change of air or surroundings will relieve a severe neuralgia. I have known obstinate cases to be cured by a prolonged residence in the mountains, with an out-of-door life and plenty of exercise. Of general remedies, iron is often a specific in the cases associated with chlorosis and anæmia. Arsenic, too, is very beneficial in these forms, and should be given in ascending doses. The value of quinine has been much overrated. It probably has no more influence than any other bitter tonic, except in the rare instances in which the neuralgia is definitely associated with malarial poisoning. Strychnine, cod-liver oil, and phosphorus are also advantageous. Of remedies for the pain, the new analgesics should first be tried—antipyrin, antifebrin, and phenacetin—for they are sometimes of service. Morphia should be given with great caution, and only after other remedies have been tried in vain. On no consideration should the patient be allowed to use the hypodermic syringe. Gelsemium is highly recommended. Of nervine stimulants, valerian and ether, which often act well together, may be given. Alcohol is a valuable, though dangerous, remedy, and should not be ordered for women. In the trifacial neuralgia nitroglycerin in large doses may be tried. Aconitia in doses of from one two-hundredth to one one-hundred-and-fiftieth of a grain may be tried. In gouty and rheumatic subjects cannabis indica and cimicifuga are recommended with the lithium salts.

Of local applications, the thermo-cautery is invaluable, particularly in zona and the more chronic forms of neuralgia. Acupuncture may be used, or aquapuncture, the injection of distilled water beneath the skin. Chloroform liniment, camphor and chloral, menthol, the oleates of morphia, atropia, and belladonna used with lanolin may be tried. Freezing over the tender point with ether spray is sometimes successful. The continuous current may be used. The sponges should be warm, and the positive pole should be placed near the seat of the pain. The strength of the current should be such as to cause a slight tingling or burning, but not pain.

The surgical treatment of intractable neuralgia embraces nerve stretching and excision. The latter is the most satisfactory, but too often the pain returns.

IX. PROFESSIONAL SPASMS; OCCUPATION NEUROSES.

The continuous and excessive use of the muscles in performing a certain movement may be followed by an irregular, involuntary spasm or cramp, which may completely check the performance of the action. The condition is found most frequently in writers, hence the term writer's cramp or scrivener's palsy; but it is also common in piano and violin players and in telegraph operators. The spasms occur in many other persons, such as milkmaids, weavers, and cigarette-rollers.

The most common form is writer's cramp, which is much more frequent in men than in women. Of 75 cases of impaired writing power reported by Poore, all of the instances of undoubted writer's cramp were in men. Morris J. Lewis states that in this country, in the telegrapher's cramp, women, who are employed a great deal in telegraphy, are much less frequently affected (only 4 out of 43 cases). Persons of a nervous temperament are more liable to the disease. Occasionally it follows slight injury.

Gowers states that in a majority of the cases a faulty method of writing has been employed, using either the little finger or the wrist as the fixed point. Persons who write from the middle of the forearm or from the elbow are rarely affected.

No anatomical changes have been found. The most reasonable explanation of the disease is that it results from a deranged action of the nerve centres presiding over the muscular movements involved in the act of writing, a condition which has been termed irritable weakness. "The education of centres which may be widely separated from each other for the performance of any delicate movement is mainly accomplished by lessening the lines of resistance between them, so that the movement, which was at first produced by a considerable mental effort, is at last executed almost unconsciously. If, therefore, through prolonged excitation, this

lessened resistance be carried too far, there is an increase and irregular discharge of nerve energy, which gives rise to spasm and disordered movement. According to this view, the muscular weakness is explained by an impairment of nutrition accompanying that of function, and the diminished faradic excitability by the nutritional disturbance descending the motor nerves." (Gay.)

Symptoms.—These may be described under five heads (Lewis).

(*a*) *Cramp* or *Spasm.*—This is often an early symptom and most commonly affects the forefinger and thumb; or there may be a combined movement of flexion and adduction of the thumb, so that the pen may be twisted from the grasp and thrown to some distance. Weir Mitchell has described a lock-spasm, in which the fingers become so firmly contracted upon the pen that it cannot be removed.

(*b*) *Paresis* and *Paralysis.*—This may occur with the spasm or alone. The patient feels a sense of weakness and debility in the muscles of the hand and arm and holds the pen feebly. Yet in these circumstances the grasp of the hand may be strong and there may be no paralysis for ordinary acts.

(*c*) *Tremor.*—This is most commonly seen in the forefinger and may be a premonitory symptom of atrophy. It is not an important symptom, and is rarely sufficient to produce disability.

(*d*) *Pain.*—Abnormal sensations, particularly a tired feeling in the muscles, are very constantly present. Actual pain is rare, but there may be irregular shooting pains in the arm. Numbness or soreness may exist. If, as sometimes happens, a subacute neuritis develops, there may be pain over the nerves and numbness or tingling in the fingers.

(*e*) *Vasomotor Disturbances.*—These may occur in severe cases. There may be hyperæsthesia. Occasionally the skin becomes glossy, or there is a condition of local asphyxia resembling chilblains. In attempting to write, the hand and arm may become flushed and hot and the veins increased in size. Early in the disease the electrical reactions are normal, but in advanced cases there may be diminution of faradic and sometimes increase in the galvanic irritability.

Diagnosis.—A well-marked case of writer's cramp or palsy could scarcely be mistaken for any other affection. Care must be taken to exclude the existence of any cerebro-spinal disease, such as progressive muscular atrophy or hemiplegia. The physician is sometimes consulted by nervous persons who fancy they are becoming subject to the disease and complain of stiffness or weakness without displaying any characteristic features.

Prognosis.—The course of the disease is usually chronic. If taken in time and if the hand is allowed perfect rest, the condition may improve rapidly, but too often there is a strong tendency to recurrence. The patient may learn to write with the left hand, but this also may after a time be attacked.

Treatment.—Various prophylactic measures have been advised. As mentioned, it is important that a proper method of writing be adopted. Gowers suggests that if all persons wrote from the shoulder writer's cramp would practically not occur. Various devices have been invented for relieving the fatigue, but none of them are very satisfactory. The use of the type-writer has diminished very much the frequency of scrivener's palsy. Rest is essential. No measures are of value without this. Massage and manipulation, when combined with systematic gymnastics, give the best results. Poore recommends the galvanic current applied to the muscles, which are at the same time rhythmically exercised.

The nutrition of the patients is apt to be much impaired, and cod-liver oil, strychnia, and other tonics will be found advantageous. Local applications are of little benefit. Tenotomy and nerve-stretching have been abandoned.

X. TETANY.

Definition.—An affection characterized by peculiar tonic spasms, either paroxysmal or continued, of the extremities.

Etiology.—The disease occurs under very different conditions. Four varieties may be recognized.

(*a*) Epidemic tetany, also known as rheumatic tetany. In certain parts of the continent of Europe the disease has prevailed widely, particularly in the winter season. Von Jaksch, who has described an epidemic form occurring in young men of the working classes, sometimes with slight fever, regards the disease as infectious. This form is acute, lasting only two or three weeks and rarely proving fatal.

(*b*) A majority of the cases are found in association with debility following lactation and chronic diarrhœa, or in the malnutrition of rickets. From its occurrence in nursing women Trousseau called it nurse's contracture. It may also occur during pregnancy. It has been found as a sequence of the acute fevers, and in some typhoid epidemics many cases have occurred.

(*c*) Tetany may follow removal of the thyroid gland. Thirteen cases, for example, followed seventy-eight operations on enlarged thyroid in Billroth's clinic, and six of them proved fatal. James Stewart has reported an instance in which with the tetany there were symptoms of myxœdema, and no trace of the thyroid gland. Removal of the thyroid in dogs has also been followed by tetany.

(*d*) And, lastly, there is a form of fatal tetany which is associated with dilatation of the stomach, particularly after the organ has been washed out. A case has been reported in this country by F. T. Miles.

On this continent tetany is an extremely rare disease. In the discussion on Stewart's case at the Association of American Physicians, Washington, 1889, Weir Mitchell stated that he had seen but two instances in

his long and varied experience, while Pepper had seen but one case, and that was in a child.

The nature of the disease is unknown, but it probably depends upon the action of some toxic agent on the motor-nerve cells.

Symptoms.—In cases associated with general debility or in children with rickets the spasm is limited to the hands and feet. The fingers are bent at the metacarpo-phalangeal joint, extended at the terminal joints, pressed close together, and the thumb is contracted in the palm of the hand. The wrist is flexed, the elbows are bent, and the arms are folded over the chest. In the lower limbs the feet are extended and the toes adducted. The muscles of the face and neck are less commonly involved, but in severe cases there may be trismus, and the angles of the mouth are drawn out. The skin of the hands and feet is sometimes tense and œdematous. The spasms are usually paroxysmal and last for a variable time. In children the attack may pass off in a few hours. In some of the severer chronic cases in adults the stiffness and contracture may continue or even increase for many days, and the attack may last as long as two weeks. In the acute cases the temperature may be elevated and the pulse quickened. In the severe paroxysms there may be involvement of the muscles of the back and of the thorax, inducing dyspnœa and cyanosis. Two additional features, valuable in diagnosis, are present. The irritability of the nerves is enormously increased both during the period of tetany and subsequently. Thus a minimal strength of current necessary to produce a contracture during the quiescent period is sufficient during the attack to cause a distinct tetanic contraction. The second point is the so-called Trousseau's phenomenon: pressure on the larger arteries, sometimes on the nerve trunk, will excite the spasm, which continues while the pressure is kept up.

Diagnosis.—The disease is readily recognized. It is a mistake to call instances of carpo-pedal spasm of children true tetany. It is common to find in rickety children or in cases of severe gastro-intestinal catarrh a transient spasm of the fingers or even of the arms. By many authors these are considered cases of mild tetany, and there are all grades in rickety children between the simple carpo-pedal spasm and the condition in which the four extremities are involved; but it is well, I think, to limit the term tetany to the severer affection.

With true tetanus the disease is scarcely ever confounded, as the commencement of the spasm in the extremities, the attitude of the hands, and the etiological factors are very different. Hysterical contractures are usually unilateral.

Except in the cases associated with dilated stomach and those which follow thyroidectomy the prospect of recovery is good.

Treatment.—In the case of children the condition with which the tetany is associated should be treated. Baths and cold sponging are recommended and often relieve the spasm as promptly as in child-crowing

Bromide of potassium may be tried. In severe cases chloroform inhalations may be given. Massage, electricity, and the spinal ice-bag have also been used with success. Cases, however, may resist all treatment, and the spasms recur for many years.

XI. HYSTERIA.

Definition.—A state in which ideas control the body and produce morbid changes in its functions (Möbius).

Etiology.—The affection is most common in women, and usually appears first about the time of puberty, but the manifestations may continue until the menopause, or even until old age. Men and boys, however, are by no means exempt, and of late years hysteria in the male has attracted much attention. It occurs in all races, but is much more prevalent, particularly in its severer forms, in members of the Latin race. In this country the milder grades are common, but the graver forms are rare in comparison with the frequency with which they are seen in France.

Of predisposing causes, two are important—heredity and education. The former acts by endowing the child with a mobile, abnormally sensitive nervous organization. We see cases most frequently in families with marked neuropathic tendencies, the members of which have suffered from neuroses of various sorts. Education at home too often fails to inculcate habits of self-control. A child grows to girlhood with an entirely erroneous idea of her relations to others, and accustomed to have every whim gratified and abundant sympathy lavished on every woe, however trifling, she reaches womanhood with a moral organization unfitted to withstand the cares and worries of every-day life. At school, between the ages of twelve and fifteen, the most important period in her life, when the vital energies are absorbed in the rapid development of the body, she is often cramming for examinations and cooped in close school-rooms for six or eight hours daily. The result too frequently is an active, bright mind in an enfeebled body, ill adapted to subserve the functions for which it was framed, easily disordered, and prone to react abnormally to the ordinary stimuli of life. Among the more direct influences are emotions of various kinds, fright occasionally, more frequently love affairs, grief, and domestic worries. Physical causes less often bring on hysterical outbreaks, but they may follow directly upon an injury or develop during the convalescence from an acute illness or be associated with disease of the generative organs. The name *hysteria* indicates how important was believed to be the part played by the uterus in the causation of the disease. Opinions differ a good deal on this question, but undoubtedly in many cases there are ovarian and uterine disorders the rectification of which sometimes cures the disease. Sexual excess, particularly masturbation, is an important factor, both in girls and boys.

Symptoms.—A useful division is into the convulsive and non-convulsive varieties.

Convulsive Hysteria.—(*a*) *Minor Forms.*—The attack most commonly follows emotional disturbance. It may set in suddenly or be preceded by symptoms, called by the laity "hysterical," such as laughing and crying alternately, or a sensation of constriction in the neck, or of a ball rising in the throat—the *globus hystericus*. Sometimes, preceding the convulsive movements, there may be painful sensations arising from the pelvic, abdominal, or thoracic regions. From the description these sensations resemble auræ. They become more intense with the rising sensation of choking in the neck and difficulty in getting breath, and the patient falls into a more or less violent convulsion. It will be noticed that the fall is not sudden, as in epilepsy, but the subject falls, as a rule, easily, often picking a soft spot, like a sofa or an easy chair, and in the movements apparently exercises care to do herself no injury. Yet at the same time she appears to be quite unconscious. The movements are clonic and disorderly, consisting of to-and-fro motion of the trunk or pelvic muscles, and the head and arms are thrown about in an irregular manner. The paroxysm after a few minutes slowly subsides, then the patient becomes emotional, and gradually regains consciousness. When questioned the patient may confess to having some knowledge of the events which have taken place, but, as a rule, has no accurate recollection. During the attack the abdomen may be much distended with flatus, and subsequently a large amount of clear urine may be passed. These attacks vary greatly in character. There may be scarcely any movements of the limbs, but after a nerve storm the patient sinks into a torpid, semi-unconscious condition, from which she is roused with great difficulty. In some cases from this state the patient passes into a condition of catalepsy.

(*b*) *Major Forms; Hystero-epilepsy.*—This condition has been specially studied by Charcot and his pupils. Typical instances passing through the various phases are very rare in this country. The attack is initiated by certain prodromata, chiefly minor hysterical manifestations, either foolish or unseemly behavior, excitement, sometimes dyspeptic symptoms with tympanites, or frequent micturition. Areas of hyperæsthesia may at this time be marked, the so-called hysterogenic spots so elaborately described by Richet. These are usually symmetrical and situated over the upper dorsal vertebra, and in front in a series of symmetrically placed spots on the chest and abdomen, the most marked being those in the inguinal regions over the ovaries. Painful sensations or a feeling of oppression and a *globus* rising in the throat may be complained of prior to the onset of the convulsion, which, according to French writers, has four distinct stages: (1) Epileptoid condition, which closely simulates a true epileptic attack with tonic spasm (often leading to opisthotonos), grinding of the teeth, congestion of the face, followed by clonic convulsions, gradual relaxation, and coma. This attack lasts rather longer than a true epi-

leptic attack. (2) Succeeding this is a period which Charcot has termed *clownism*, in which there is an emotional display and a remarkable series of contortions or of cataleptic poses. (3) Then in typical cases there is a stage in which the patient assumes certain attitudes expressive of the various passions—ecstasy, fear, beatitude, or erotism. (4) Finally consciousness returns and the patient enters upon a stage in which she may display very varied symptoms, chiefly manifestations of a delirium with the most extraordinary hallucinations. Visions are seen, voices heard, and conversations held with imaginary persons. In this stage patients will relate with the utmost solemnity imaginary events, and make extraordinary and serious charges against individuals. This sometimes gives a grave aspect to these seizures, for not only will the patient at this stage make and believe the statements, but when recovery is complete the hallucination sometimes persists. We seldom see in this country attacks having this orderly sequence. Much more commonly the convulsions succeed each other at intervals for several days in succession. Here is a striking difference between hystero-epilepsy and true epilepsy. In the latter the status epilepticus, if persistent, is always serious, associated with fever, and frequently fatal, while in hystero-epilepsy attacks may recur for days without special danger to life. After an attack of hystero-epilepsy the patient may sink into a state of trance or lethargy, in which she may remain for days.

Non-convulsive Forms.—So complex and varied is the clinical picture of hysteria that various manifestations are best considered according to the systems which are involved.

(1) **Disorders of Motion.**—(*a*) *Paralyses.*—These may be hemiplegic, paraplegic, or monoplegic. Hysterical diplegia is extremely rare. The paralysis either sets in abruptly or gradually, and may take weeks to attain its full development. *There is no type or form of organic paralysis which may not be simulated in hysteria.* According to Weir Mitchell, the hemiplegias are most frequent in the ratio of four on the left to one on the right side. The face is not affected; the neck may be involved, but the leg suffers most. Sensation is either lessened or lost on the affected side. The hysterical paraplegia is more common than hemiplegia. The loss of power is not absolute; the legs can usually be moved, but do not support the patient. The reflexes may be increased, though the knee-jerk is often normal. A spurious ankle clonus may sometimes be present. The feet are usually extended and turned inward in the equino-varus position. The muscles do not waste and the electrical reactions are normal. Other manifestations, such as paralysis of the bladder or aphonia, are usually associated with the hysterical paraplegia. Hysterical monoplegias may be facial, crural, or brachial. A condition of ataxia sometimes occurs with paresis. The incoördination may be a marked feature, and there are usually sensory manifestations.

(*b*) *Contractures and Spasms.*—An extraordinary variety of spas-

modic affections occurs in hysteria, of which the most common are the following: The hysterical contractures may attack almost any group of voluntary muscles and be of the hemiplegic, paraplegic, or monoplegic type. They may come on suddenly or slowly, persist for months or years, and disappear rapidly. The contracture is most commonly seen in the arm, which is flexed at the elbow and wrist, and the fingers tightly grasp the thumb in the palm of the hand; more rarely the terminal phalanges are hyperextended as in athetosis. It may occur in one or in both legs, more commonly the former. The ankle clonus is present; the foot is inverted and the toes are strongly flexed. These cases may be mistaken for lateral sclerosis and the difficulty in diagnosis may really be very great. The spastic gait is very typical, and with the exaggerated knee-jerk and ankle clonus the picture may be characteristic. In 1879 I frequently showed such a case at the Montreal General Hospital as a typical example of lateral sclerosis. The condition persisted for more than eighteen months and then disappeared completely. Other forms of contracture may be in the muscles of the hip, shoulder, or neck; more rarely in those of the jaws —hysterical trismus—or in the tongue. Remarkable indeed are the local contractures in the diaphragm and abdominal muscles, producing a phantom tumor, in which just below and in the neighborhood of the umbilicus is a firm, apparently solid growth. According to Gowers, this is produced by relaxation of the recti and a spasmodic contraction of the diaphragm, together with inflation of the intestines with gas and an arching forward of the vertebral column. They are apt to occur in middle-aged women about the menopause, and are frequently associated with the symptoms of spurious pregnancy—*pseudo-cyesis*. The resemblance to a tumor may be striking, and I have known skilful diagnosticians to be deceived. The only safeguard is to be found in complete anæsthesia, when the tumor entirely disappears. Some years ago I went by chance into the operating-room of a hospital and found a patient on the table under chloroform and the surgeon prepared to perform ovariotomy. The tumor, however, had completely disappeared with full anæsthesia. Mitchell has reported an instance of a phantom tumor in the left pectoral region just above the breast, which was tender, hard, and dense.

Clonic spasms are more common in hysteria in this country than contractures. The following are the important forms: *Rhythmic hysterical spasm*. This, unfortunately, is sometimes known as rhythmic chorea or hysterical chorea. The movements may be of the arm, either flexion and extension, or, more rarely, pronation and supination. Clonic contractions of the sterno-cleido-mastoid or of the muscles of the jaws or of the rotatory muscles of the head may produce rhythmic movements of these parts. The spasm may be in one or both psoas muscles, lifting the leg in a rhythmic manner eight or ten times in a minute. In other instances the muscles of the trunk are affected, and every few moments there is a bowing movement—salaam convulsions—or the muscles of the back may

contract, causing strong arching of the vertebral column and retraction of the head. These movements may often alternate, as in a case in my wards, in which the patient on fine days had regular salaam convulsions, while on wet days the rhythmic spasm was in the muscles of the back and neck. Mitchell has described a rotatory spasm in which the patient rotated involuntarily, usually to the left. More unusual cases are those in which the contractions closely simulate paramyoclonus multiplex. A characteristic example of this was recently at my clinic. Hysterical athetosis is a rare form of spasm. *Tremor* may be a pure hysterical manifestation, occurring either alone or with paralysis and contracture. It most commonly involves the hands and arms; more rarely the head and legs. The movements are small and quick. Volitional or intentional tremor may exist, simulating closely the movements of insular sclerosis. Buzzard states that many instances of this disease in young girls are mistaken for hysteria.

(2) **Disorders of Sensation.**—*Anæsthesia* is most common, and usually confined to one half of the body. It may not be noticed by the patient. Usually it is accurately limited to the middle line and involves the mucous surfaces and deeper parts. The conjunctiva, however, is often spared. There may be hemianopia. This symptom may come on slowly or follow a convulsive attack. Sometimes the various sensations are dissociated and the anæsthesia may be only to pain and to touch. The skin of the affected side is usually pale and cool, and a pin-prick may not be followed by blood. With the loss of feeling there may be loss of muscular power. Curious trophic changes may be present, as in an interesting case of Weir Mitchell's, in which there was unilateral swelling of the hemiplegic side.

A phenomenon to which much attention has been paid is that of transference. By metallotherapy, the application of certain metals, the anæsthesia or analgesia can be transferred to the other side of the body. It has been shown, however, that this phenomenon may be caused by the electro-magnet and by wood and various other agents, and is probably entirely a mental effect. The subject has no practical importance, but it remains an interesting and instructive chapter in Gallic medical history.

Hyperæsthesia.—Increased sensitiveness and pains occur in various parts of the body. One of the most frequent complaints is of pain in the head, usually over the sagittal suture, less frequently in the occiput. This is described as agonizing, and is compared to the driving of a nail into the part; hence the name *clavus hystericus*. Neuralgias are common. Hyperæsthetic areas, the hysterogenic points, exist on the skin of the thorax and abdomen, pressure upon which may cause minor manifestations or even a convulsive attack. Increased sensitiveness exists in the ovarian region, but is not peculiar to hysteria. Pain in the back is an almost constant complaint of hysterical patients. The sensitiveness may be limited to certain spinous processes, or it may be diffuse. In hysterical women the pains in the abdomen may simulate those of gastralgia and of gastric

ulcer, or the condition may be almost identical with that of peritonitis; more rarely the abdominal pains closely resemble those of appendix disease.

Special Senses.—Disturbances of taste and smell are not uncommon and may cause a good deal of distress. Of ocular symptoms, retinal hyperæsthesia is the most common, and the patients always prefer to be in a darkened room. Retraction of the field of vision is common and usually follows a convulsive seizure. It may persist for years. The color perception may be normal even with complete anæsthesia, and in this country the achromatopsia does not seem to be nearly so common an hysterical manifestation as in Europe. Hysterical deafness may be complete and may alternate or come on at the same time with hysterical blindness.

(3) **Visceral Manifestations.**—*Respiratory Apparatus.*—Of disturbances in the respiratory rhythm, the most frequent, perhaps, is an exaggeration of the deeper breath, which is taken normally every fifth or sixth inspiration, or there may be a "catching" breathing, such as is seen when cold water is poured over a person. Hysterical dyspnœa is readily recognized, as there is no special distress and the pulse is usually normal. I have met with a remarkable case following trauma in which the respirations rose above one hundred and thirty in the minute. Among laryngeal manifestations aphonia is the most frequent and may persist for months or even years without other special symptoms of the disease. Spasm the muscles may occur with violent inspiratory efforts and great distr and may even lead to cyanosis. Hiccough, or sounds resembling it, may be present for weeks or months at a time. Among the most remarkable of the respiratory manifestations are the hysterical cries. These may mimic the sounds produced by animals, such as barking, mewing, or grunting, and in France epidemics of them have been repeatedly observed. Extraordinary cries may be produced, either inspiratory or expiratory. I saw at Wagner's clinic at Leipsic a girl of thirteen or fourteen, who had for many weeks given utterance to a remarkable inspiratory cry somewhat like the whoop of whooping-cough, but so intense that it was heard at a long distance. It was incessant, and the girl was worn to a skeleton. Attacks of gaping, yawning, and sneezing may also occur.

The hysterical cough is a frequent symptom, particularly in young girls. It may occur in paroxysms, but is often a dry, persistent, croaking cough, extremely monotonous and unpleasant to hear. Sir Andrew Clark has called attention to a loud, barking cough occurring about the time of puberty, chiefly in boys belonging to neurotic families. The attacks, which last about a minute, recur frequently.

There is a peculiar form of hæmoptysis which may be very deceptive and lead to the diagnosis of pulmonary disorders. Wagner describes the sputum as a pale-red fluid—not so bright in color as in ordinary hæmoptysis, and on settling presents a reddish-brown sediment. It contains particles of food, pavement epithelium, red corpuscles, and micrococci, but

no cylindrical or ciliated epithelium. It probably comes from the mouth or pharynx.

Digestive System.—Disturbed or depraved appetite, dyspepsia, and gastric pains are common in hysterical patients. The patient may have difficulty in swallowing the food, apparently from spasm of the gullet. There are instances in which the food seems to be expelled before it reaches the stomach. In other cases there is incessant gagging. In the hysterical vomiting the food is regurgitated without much effort and without nausea. This feature may persist for years without great disturbance of nutrition. The most striking and remarkable digestive disturbance in hysteria is the *anorexia nervosa* described by Sir William Gull. "To call it loss of appetite—anorexia—but feebly characterizes the symptom. It is rather an annihilation of appetite, so complete that it seems in some cases impossible ever to eat again. Out of it grows an antagonism to food which results at last and in its worst forms in spasm on the approach of food, and this in turn gives rise to some of those remarkable cases of survival for long periods without food" (Mitchell). As this goes on there may be an extreme degree of muscular restlessness, so that the patients wander about until exhausted. This feature has not been present in the cases which have come under my observation. Nothing more pitiable is to be seen in practice than an advanced case of this sort. It is usually in a young girl, sometimes as early as the eleventh or twelfth, more commonly between the fifteenth and twentieth years. The emaciation is frightful, and scarcely exceeded by that of cancer of the œsophagus. The patient finally takes to bed, and in extreme cases lies upon one side with the thighs and legs flexed, and contractures may occur. Food is either not taken at all or only upon urgent compulsion. The skin becomes wasted, dry, and covered with bran-like scales. No food may be taken for several weeks at a time, and attempts to feed may be followed by severe spasms. Although the condition looks so alarming, these cases, when removed from their home surroundings and treated by Weir Mitchell's method, sometimes recover in a remarkable way. Death, however, may follow with extreme emaciation. In a fatal case recently under my care the girl weighed only forty-nine pounds. No lesions were found post mortem.

Among intestinal symptoms flatulency is one of the most distressing, and is usually associated with the condition of peristaltic unrest (Küssmaul). Frequent discharges of fæces may be due to disturbance in either the small or large bowel. An obstinate form of diarrhœa is found in some hysterical patients, which proves very intractable and is associated especially with the taking of food. It seems an aggravated form of the looseness of bowels to which so many nervous people are subject on emotion or the tendency which some have to diarrhœa immediately after eating. An entirely different form is that produced by what Mitchell calls the irritable rectum, in which scybala are passed frequently during the day, sometimes with great violence. Constipation is more frequent, however, and may be

due to a loss of power in the muscles of the bowel or in the abdominal muscles. In extreme cases the bowels may not be moved for two or three weeks, leading to great accumulation of fæces. Other disturbances are ano-spasm or intense pain in the rectum apart from any fissure.

Cardio-vascular.—Rapid action of the heart on the slightest emotion, with or without the subjective sensation of palpitation, is often a source of great distress. A slow pulse is less frequent. Pains about the heart may simulate angina, the so-called hysterical or pseudo-angina, which has already been considered. Flushes in various parts are among the most common symptoms, and may be seen in the head, back, hands, or feet. Sweating occasionally occurs.

Among the more remarkable vaso-motor phenomena are the so-called stigmata or hæmorrhages in the skin, such as were present in the celebrated case of Louise Lateau. In many cases these are undoubtedly fraudulent, but if, as appears credible, such bleeding may exist in the hypnotic trance, there seems no reason to doubt its occurrence in the trance of prolonged religious ecstasy.

Joint Affections.—To Sir Benjamin Brodie and Sir James Paget we owe the recognition of these extraordinary manifestations of hysteria. Perhaps no single affection has brought more discredit upon the profession, for the cases are very refractory, and finally fall into the hands of a charlatan or faith-healer, under whose touch the disease may disappear at once. Usually it affects the knee or the hip, and may follow a trifling injury. The joint is usually fixed, sensitive, and swollen. The surface may be cool, but sometimes the local temperature is increased. To the touch it is very sensitive and movement causes great pain. In protracted cases the muscles about the joint are somewhat wasted, and in consequence it looks larger. The pains are often nocturnal, at which time the local temperature may be much increased. While, as a rule, neuromimetic joints yield to proper management, there are interesting instances in the literature in which organic change has succeeded the functional disturbance. In the remarkable case reported in Weir Mitchell's lectures, the hysterical features were pronounced, and, on account of the chronicity, the disease of the knee-joint was considered organic by such an authority as Billroth. Sands operated and found the joint surfaces normal, and the thickening to be due to non-tuberculous inflammatory products outside the capsule.

Mental Symptoms.—The psychical condition of an hysterical patient is always abnormal, and the disease occupies the ill-defined territory between sanity and insanity. In a large number of cases the patients are really insane, particularly in the perversion witnessed in the moral sphere. Not the slightest dependence can be placed upon their statements, and they will for months or years deceive friends, relatives, and physician. This appears to result partly, but not wholly, from a morbid craving for sympathy. It is really due to an entire unhinging of the moral nature.

Hysterical patients may become insane and display persistent hallucinations and delirium, alternating perhaps with emotional outbursts of an aggravated character. For weeks or months they may be confined to bed, entirely oblivious to their surroundings, with a delirium which may simulate that of delirium tremens, particularly in being associated with loathsome and unpleasant animals. The nutrition may be maintained, but in these cases there is always a very heavy, foul breath. With seclusion and care recovery usually takes place within three or four months. At the onset of these attacks and during convalescence the patients must be incessantly watched, as a suicidal tendency is by no means uncommon.

Of hysterical manifestations in the higher centres that of trance is the most remarkable. This may develop spontaneously without any convulsive seizure, but more frequently, in this country at least, it follows hysteroid attacks. Catalepsy, a condition in which the limbs are plastic and remain in any position in which they are placed, may or may not be present with this condition.

The Metabolism in Hysteria.—The studies of Gilles de la Tourette and Cathelineau, under Charcot's direction, have shown that in the ordinary forms of hysteria the urine does not show quantitative or qualitative changes, but in the severe types, characterized by convulsions, etc., there are important modifications: reduction in the urates and phosphates; the ratio of the earthy to the alkaline phosphates, normally 1:3, is 1:2, or even 1:1. The urine is also reduced in amount. They think that these changes might sometimes serve to differentiate convulsive hysteria from epilepsy, in which there is always an increase in the solid constituents after a seizure.

Hysterical Fever.—In hysteria the temperature, as a rule, is normal. The cases with fever may be grouped as follows: (*a*) Instances in which the fever is the sole manifestation. These are rare, but I have seen at least two cases in which the chronic course, the retention of the nutrition, and the entirely negative condition of the organs left no other diagnosis possible. In a case recently under observation the patient has had for four or five years an afternoon rise of temperature, reaching usually to 102° or 103°. She was well nourished and presented no pronounced hysterical symptoms, but there was a marked neurotic history on one side and a form of interrupted sighing respiration so often seen in hysteria.

(*b*) Cases of hysterical fever with spurious local manifestations. These are very troublesome and deceptive cases. The patient may be suddenly taken ill with pain in various regions and elevation of temperature. The case may simulate meningitis. There may be pain in the head, vomiting, contracted pupils, and retraction of the neck—symptoms which may persist for weeks—and some anomalous manifestation during convalescence may alone indicate to the physician that he has had to deal with a case of hysteria, and has not, as he perhaps flattered himself, cured a case of meningitis. Mary Putnam Jacobi, in a recent article on hysterical fever,

mentions a case in the service of Cornil which was admitted with dyspnœa, slight cyanosis, and a temperature at 39° C. The condition proved to be hysterical. There is also an hysterical pseudo-phthisis with pain in the chest, slight fever, and the expectoration of a blood-stained mucus. The cases of hysterical peritonitis may also show fever. Only by incessant watchfulness can mistakes be prevented in these cases.

(*c*) *Hysterical Hyperpyrexia.*—It is a suggestive fact that the cases of paradoxical temperatures reported of late years, in which the thermometer has registered 112° to 120° or more, have been in women. Fraud has been practised in some of these, but in others the high fever has been associated with neurotic features and may really have been of an hysterical character.

Diagnosis.—Inquiry into the occurrence of previous manifestations and the mental conditions may give important information. These questions, as a rule, should not be asked the mother, who of all others is least likely to give satisfactory information about the patient's condition. The occurrence of the globus hystericus, of emotional attacks, of weeping and crying, are always suggestive. The points of difference between the convulsive attacks and true epilepsy were referred to in their description, and as a rule little difficulty is experienced in distinguishing between the two conditions. The hysterical paralyses are very variable and apt to be associated with anæsthesia. The contractures may at times be very deceptive, but the occurrence of areas of anæsthesia, of retraction of the visual field, and the development of minor hysterical manifestations, give valuable indications. The contractures disappear under full anæsthesia. Special care must be taken not to confound the spastic paraplegia of hysteria with lateral sclerosis.

The visceral manifestations are usually recognized without much difficulty. The practitioner has constantly to bear in mind the strong tendency in hysterical patients to practise deception.

Treatment.—The prophylaxis in hysteria may be gathered from the remarks on the relation of education to the disease. The successful treatment of hysteria demands qualities possessed by few physicians. The first element is a due appreciation of the nature of the disease on the part of the physician and friends. It is pitiable to think of the misery which has been inflicted on these unhappy victims by the harsh and unjust treatment which has resulted from false views of the nature of the trouble; on the other hand, worry and ill-health, often the wrecking of mind, body, and estate, are entailed upon the near relatives in the nursing of a protracted case of hysteria. The minor manifestations, attacks of the vapors, the crying and weeping spells, are not of much moment and rarely require treatment. The physical condition should be carefully looked into and the mode of life regulated so as to insure system and order in everything. A congenial occupation offers the best remedy for many of these manifestations. Any functional disturbance should be at-

tended to and a course of tonics prescribed. Special attention should be paid to the action of the bowels.

Valerian and asafœtida are often of service. For the pains in various parts, particularly in the back, the thermo-cautery and static electricity will be found invaluable. Morphia should be withheld. In the convulsive seizures, particularly in the minor forms, it is often best, after settling the patient comfortably, to leave her. When she comes to, and finds herself alone and without sympathy, the attacks are less likely to be repeated. There is, as a rule, no cure for the hysterical manifestations of women, otherwise in good health, who are, as Mitchell says, "fat and ruddy, with sound organs and good appetites, but ever complain of pains and aches, and ever liable on the least emotional disturbance to exhibit a quaint variety of hysterical phenomena."

To treat hysteria as a physical disorder is, after all, radically wrong. It is essentially a mental and emotional anomaly, and the important element in the treatment is moral control. At home, surrounded by loving relatives who misinterpret entirely the symptoms and have no appreciation of the nature of the disease, the severer forms of hysteria can rarely be cured. The necessary control is impossible; hence the special value of the method introduced by Weir Mitchell, which is particularly applicable to the advanced cases which have become chronic and bedridden. The treatment consists in isolation, rest, diet, massage, and electricity. Separation from friends and sympathetic relatives must be absolute, and can rarely, if ever, be obtained in the individual's home. An essential element in the treatment is an intelligent nurse. No small share of the success which has attended the author of this plan h[illegible]een due to the fact that he has persistently chosen as his allies bright, intelligent women. The details of the plan are as follows: The patient is confined [illegible] bed and not allowed to get up, nor, at first, in aggravated cases, to read, write, or even to feed herself. Massage is used daily, at first for twenty minutes or half an hour, subsequently for a longer period. It is essential as a substitute for exercise. The induction current is applied to the various muscles and to the spine. Its use, however, is not so essential as that of massage. The diet may at first be entirely of milk, four ounces every two hours. It is better to give skimmed milk, and it may be diluted with soda water or barley water and, if necessary, peptonized. After a week or ten days the diet may be increased, the amount of milk still being kept up. A chop may be given at midday, a cup of coffee or cocoa with toast or bread and butter or a biscuit with the milk. The patients usually fatten rapidly as the solid food is added, and with the gain there is, as a rule, a diminution or cessation of the nervous symptoms. The milk is the essential element in the diet, and is itself amply sufficient.

The remarkable results obtained by this method are now universally recognized. The plan is more applicable to the lean than to fat, flabby hysterical patients. Not only is it suitable for the more obstinate varie-

ties of hysteria with bodily manifestations, but in the cases with mental symptoms the seclusion and separation from relatives and friends are particularly advantageous. In the hysterical vomiting Debove's method of forced feeding may be used with benefit. For the innumerable minor manifestations of hysteria and for the simulations the indications for treatment are usually clear. Of late, hypnotism has been extensively used in the treatment of hysteria. Occasionally in cases of hysterical contractions or paralysis it is of benefit, but any one who has seen the development of this method as practised at present in France must feel that it is a two-edged sword and that the constant repetition in the same patient is fraught with danger. In the cases which we have tried here the success has not been marked.

XII. NEURASTHENIA.

Definition.—A condition of weakness or exhaustion of the nervous system.

The term, invented by Beard, covers an ill-defined, motley group of symptoms, which may be either general and the expression of derangement of the entire system, or local, limited to certain organs; hence the terms cerebral, spinal, cardiac, and gastric neurasthenia. In certain respects it is the physical counterpart of insanity. As the essential feature in the latter condition is the abnormal response to stimuli, from within or without, upon the higher centres presiding over the mind, so neurasthenia appears to be the expression of a morbid, unhealthy reaction to stimuli acting on the nervous centres which preside over the functions of organic life. No hard and fast line can be drawn between neurasthenia and certain mental states, particularly hysteria and hypochondria.

Etiology.—Although the causes are apparently varied, they may be grouped as hereditary and acquired.

(*a*) *Hereditary.*—We do not all start in life with the same amount of nerve capital. Parents who have been the subjects of nervous complaints or of mental troubles transmit to their children an organization which is defective in what, for want of a better term, we must call "nerve force." Such individuals start handicapped, and furnish a considerable proportion of our neurasthenic patients. So long as they are content to transact a moderate business with their life capital, all may go well, but there is no reserve, and in the emergencies which constantly arise in the exigencies of modern life these small capitalists go under and come to us as bankrupts.

(*b*) *Acquired.*—The functions, though perverted most readily in persons who have inherited a feeble organization, may also be damaged by exercise which is excessive in proportion to the strength—i. e., by strain. The cares and anxieties attendant upon the gaining of a livelihood may be borne without distress, but in many persons the strain becomes excess-

ive and is first manifested as *worry*. The individual loses the distinction between essentials and non-essentials, trifles cause annoyance, and the entire organism reacts with unnecessary readiness to slight stimuli, and is in a state which the older writers called irritable weakness. If such a condition be taken early and the patient given rest, the balance is quickly restored. In this group may be placed a large proportion of the neurasthenics which we see in this country, particularly among business men. Other causes more subtle, yet potent, and less easily dealt with, are the worries attendant upon love affairs, religious doubts, and the sexual passion.

Symptoms.—These are extremely varied, and may be general or localized; more often a combination of both. The appearance of the patient is suggestive, sometimes characteristic, but difficult to describe. Loss of weight and slight anæmia may be present. The physical debility may reach a high grade and the patient may be confined to bed. Mentally the patients are usually low-spirited and despondent, in women frequently emotional.

The local symptoms may dominate the situation, in which case the clinical picture is of the so-called cerebral or spinal neurasthenia. Other local forms are cardio-vascular, gastric, and sexual.

In the cerebral form the symptoms are chiefly connected with an inability to perform the ordinary mental work. Thus a row of figures cannot be correctly added, the dictation or the writing of a few letters is a source of the greatest worry, the transaction of petty details in business is a painful effort, and there is loss of power of fixed attention. With this condition there may be no headache, the appetite may be good, and the patient may sleep well. As a rule, however, there are sensations of fulness and weight or flushes, if not actual headache. Sleeplessness is a frequent concomitant, and may be the first manifestation. Some of these patients are good-tempered and cheerful, but a majority are moody, irritable, and depressed.* The special senses may be disturbed, particularly vision. An aching or weariness of the eyeballs after reading a few minutes or flashes of light are common symptoms. A difference between the pupils may be present.

When the *spinal symptoms* predominate—spinal irritation or spinal neurasthenia—in addition to many of the features just mentioned, the patients complain of weariness on the least exertion, of weakness, pain in the back, and of aching pains in the legs. There may be spots of local tenderness on the spine. Occasionally there may be disturbances of sensation, particularly a feeling of numbness and tingling, and the reflexes may be increased. The aching pain in the back or in the back of the neck is the most constant complaint in these cases. In women it is often

* For an exhaustive consideration of the mental symptoms of neurasthenia, see the Shattuck Lecture, by Cowles. Boston Medical and Surgical Journal, 1891.

impossible to say whether this condition is one of neurasthenia or hysteria.

In other cases the *cardio-vascular* symptoms are the most distressing, and may occur with only slight disturbance of the cerebro-spinal functions, though the conditions may be combined. Palpitation of the heart, irregular and very rapid action, and pains in the cardiac region are the most common symptoms. The slightest excitement may be followed by increased action of the heart, and the patients frequently have the idea that they suffer from serious disease of this organ.

Vaso-motor disturbances constitute a special feature of many cases. Flushes of heat and transient hyperæmia of the skin may be very distressing symptoms. Profuse sweating may occur, either local or general, and sometimes nocturnal. The pulse may show interesting features, owing to the extreme relaxation of the peripheral arterioles. The arterial throbbing may be everywhere visible, almost as much as in aortic insufficiency. The pulse, too, may under these circumstances have a somewhat water-hammer quality. The capillary pulse may be seen in the nails, on the lips, or on the margins of a line drawn upon the forehead, and I have on several occasions seen pulsation in the veins of the back of the hand. A characteristic symptom in some cases is the *throbbing aorta*. The epigastric pulsation may be extremely forcible and suggest the existence of abdominal aneurism. The subjective sensations associated with it may be very unpleasant, particularly when the stomach is empty.

The general features of gastro-intestinal neurasthenia have been dealt with under the section of nervous dyspepsia. The connection of these cases with dilatation of the stomach, floating kidney, and the condition which Glénard calls *enteroptosis* has already been mentioned.

Sexual neurasthenia is a condition in which there is an irritable weakness of the sexual organs manifested by nocturnal emissions, unusual depression after intercourse, and often by a distressing dread of impotence. The mental condition of these patients is most pitiable, and they fall an easy prey to quacks and charlatans of all kinds.

In all forms of neurasthenia the condition of the urine is important. Many cases are complicated with the symptoms of the condition known as lithæmia, and so marked may this be that some have indeed made a special form of lithæmic neurasthenia. Polyuria may be present, but is more common in hysteria. With disturbed digestion the urates and oxalates may be in excess.

The *diagnosis* is readily made. It is sometimes difficult to distinguish the cases from hysteria, and this is not surprising, as we cannot always differentiate the two conditions. Neurasthenia occurs chiefly in men; in fact, it is in many ways in them the equivalent of hysteria.

XIII. THE TRAUMATIC NEUROSES

(*Railway Brain and Railway Spine; Traumatic Hysteria*).

Definition.—A morbid condition following shock which presents the symptoms of neurasthenia or hysteria or of both. The condition is known as "railway brain" and "railway spine."

Erichsen regarded the condition as the result of inflammation of the meninges and cord, and gave it the name railway spine. Walton and J. J. Putnam, of Boston, were the first to recognize the hysterical nature of many of the cases,* and to Westphal's pupils we owe the name traumatic neurosis.

Etiology.—The condition follows an accident, often in a railway train, in which injury has been sustained, or succeeds a shock or concussion, from which the patient may apparently not have suffered in his body. A man may appear perfectly well for several days, or even a week or more, and then develop marked symptoms of the neurosis. Bodily shock or concussion is not necessary. The affection may follow a profound mental impression; thus, an engine driver ran over a child, and received thereby a very severe shock, subsequent to which the most pronounced symptoms of neurasthenia developed. Severe mental strain combined with bodily exposure may cause it, as in a case of a naval officer who was wrecked in a violent storm and exposed for more than a day in the rigging before he was rescued. A slight blow, a fall from a carriage or on the stairs may suffice.

Symptoms.—The cases may be divided into three groups: simple neurasthenia, cases with marked hysterical manifestations, and cases with severe symptoms indicating or simulating organic disease.

(*a*) *Simple Traumatic Neurasthenia.*—The first symptoms usually develop a few weeks after the accident, which may or may not have been associated with an actual trauma. The patient complains of headache and tired feelings. He is sleepless and finds himself unable to concentrate his attention properly upon his work. A condition of nervous irritability develops, which may have a host of trivial manifestations, and the entire mental attitude of the person may for a time be changed. He dwells constantly upon his condition, gets very despondent and low-spirited, and in extreme cases melancholia may develop. He may complain of numbness and tingling in the extremities, and in some cases of much pain in the back. The bodily functions may be well performed, though such patients usually have, for a time at least, disturbed digestion and loss in weight. The physical examination may be entirely negative. The reflexes are slightly increased, as in ordinary neurasthenia. The pupils may be unequal; the cardio-vascular changes already described in neurasthenia may be present in a marked degree. According as the symptoms are more

* See La Neurasthénie, par L. Bouveret, Paris, 1891.

spinal or more cerebral, the condition is known as railway brain or railway spine.

(2) *Cases with Marked Hysterical Features.*—Following an injury of any sort, neurasthenic symptoms, like those described above, may develop, and in addition symptoms regarded as characteristic of hysteria. The emotional element is prominent, and there is but slight control over the feelings. The patients have headache, backache, and vertigo. A violent tremor may be present, and indeed constitutes the most striking feature of the case. I have recently seen an engineer who developed subsequent to an accident a series of nervous phenomena, but the most marked feature was an excessive tremor of the entire body, which was specially manifest during emotional excitement. The most pronounced hysterical symptoms are the sensory disturbances. As first noted by Putnam and Walton, hemianæsthesia may occur as a sequence of traumatism. This is a common symptom in France, but rare in England and in this country. In a considerable number of cases of traumatic neuroses which I have seen only one presented typical hemianæsthesia. A second, more common, manifestation is limitation of the field of vision.

Remarkable disturbances may develop in some of these cases. A few months ago I saw a man who had been struck by an electric car, whose chief symptom was an extraordinary increase in the number of respirations. He was a stout, powerfully built man, and presented practically no other symptom than dyspnœa of the most extreme grade. At the time of observation his respirations were over 130 per minute, and he stated that they had been counted at over 150.

(3) *Cases in which the Symptoms suggest Organic Disease of the Brain and Cord.*—As a result of spinal concussion, without fracture or external injury, there may subsequently develop symptoms suggestive of organic disease, which may come on rapidly or at a late date. In a case reported by Leyden the symptoms following the concussion were at first slight and the patient was regarded as a simulator, but finally the condition became aggravated and death resulted. The post-mortem showed a chronic pachymeningitis, which had doubtless resulted from the accident. The cases in this group about which there is so much discussion are those which display marked sensory and motor changes. Following an accident in which the patient has not received external injury a condition of excitement may develop within a week or ten days; he complains of headache and backache, and on examination sensory disturbances are found, either hemianæsthesia or areas on the skin in which the sensation is much benumbed; or painful and tactile impressions may be distinctly felt in certain regions, and the temperature sense is absent. The distribution may be bilateral and symmetrical in limited regions or hemiplegic in type. Limitation of the field of vision is usually marked in these cases, and there may be disturbance of the senses of taste and smell. The superficial reflexes may be diminished; usually the deep reflexes are exaggerated. The

pupils may be unequal; the motor disturbances are variable. The French writers describe cases of monoplegia with or without contracture, symptoms upon which Charcot lays great stress as a manifestation of profound hysteria. The combination of sensory disturbances—anæsthesia or hyperæsthesia—with paralysis, particularly if monoplegic, and the occurrence of contractures without atrophy and with normal electrical reactions, may be regarded as distinctive of hysteria.

In rare cases following trauma and succeeding to symptoms which may have been regarded as neurasthenic or hysterical, there are organic changes which may prove fatal. That this sequence occurs is demonstrated clearly by recent post-mortem examinations. The features upon which the greatest reliance can be placed as indicating definite organic change are optic atrophy, bladder symptoms, particularly in combination with tremor, paresis, and exaggerated reflexes.

The anatomical changes in this condition have not been very definite. When death follows spinal concussion within a few days there may be no apparent lesion, but in some instances the brain or cord has shown punctiform hæmorrhages. Edes has reported four cases in which a gradual degeneration in the pyramidal tracts followed concussion or injury of the spine; but in all these cases there was marked tremor and the spinal symptoms developed early or followed immediately upon the accident. Post-mortems upon cases in which organic lesions have supervened upon a traumatic neurosis are extremely rare. Bernhardt reports an instance of a man, aged thirty-three, who in 1886 received a kick from a horse on the epigastrium and subsequently developed the symptom-complex of neurasthenia and hysteria with attacks of vertigo and great psychical depression. He afterward had more marked mental symptoms and attacks of unconsciousness. He committed suicide and the brain and cord showed a beginning multiple sclerosis in the white matter, which was possibly associated with an advanced grade of arterio-sclerosis. In a second case a man, aged forty-two, received a shock in a railway accident in July, 1884. He was rendered unconscious and had a slight injury in the buttock region. In a few weeks symptoms of traumatic neurosis developed, particularly great depression of spirits, with headache and sensory disturbances in the feet and hands. Tremor and great weakness were complained of when he attempted to work. There was no increase in the reflexes. The case was regarded as an instance of simulation and a defect in objective symptoms favored this view. Subsequently this judgment was reversed, but he did not improve. He died in January, 1889, with symptoms of cardiac dyspnœa. Macroscopically the brain and cord appeared normal. There was extreme arterio-sclerosis, particularly of the vessels of the brain and cord. In the latter there were scattered areas of degeneration in the white substance, and degeneration in the sympathetic ganglia.

I have entered somewhat fully into this question because of its extreme

importance and on account of the paucity of the observations upon cases which have subsequently developed symptoms of organic disease. Examples of it are extremely rare. So far as I know no case with autopsy has been reported in this country, nor have I seen an instance in which the clinical features pointed to an organic disease which had followed upon a traumatic neurosis.

Diagnosis.—A condition of fright and excitement following an accident may persist for days or even weeks, and then gradually pass away. The symptoms of neurasthenia or of hysteria which subsequently develop present nothing peculiar and are identical with those which occur under other circumstances. Care must be taken to avoid simulation, and, as in these cases the condition is largely subjective, this is sometimes extremely difficult. In a careful examination a simulator will often reveal himself by exaggeration of certain symptoms, particularly sensitiveness of the spine, and by increasing voluntarily the reflexes. It may require a careful study of the case to determine whether the individual is honestly suffering from the symptoms of which he complains. A still more important question in these cases is, Has the patient organic disease? The symptoms given under the first two groups of cases may exist in a marked degree and may persist for several years without the slightest evidence of organic change. It must be noted that in the two autopsies above referred to the patients were the subjects of extreme arterio-sclerosis, with which, in all probability, the areas of multiple sclerosis were associated. Hemianæsthesia, limitation of the field of vision, monoplegia with contracture, may all be present as hysterical manifestations, from which recovery may be complete. In our present knowledge the diagnosis of an organic lesion should be limited to those cases in which optic atrophy, bladder troubles, and signs of sclerosis of the cord are well marked—indications either of degeneration of the lateral columns or of multiple sclerosis.

Prognosis.—A majority of patients with traumatic hysteria recover. In railway cases, so long as litigation is pending and the patient is in the hands of lawyers the symptoms usually persist. Settlement is often the starting point of a speedy and perfect recovery. I have known return to health after the persistence of the most aggravated symptoms with complete disability of from three to five years' duration. On the other hand, there are a few cases in which the symptoms persist even after the litigation has been closed; the patient goes from bad to worse and psychoses develop, such as melancholia, dementia, or occasionally progressive paresis. And, lastly, in extremely rare cases, organic lesions may develop as a sequence of the traumatic neurosis.

The function of the physician acting as medical expert in these cases consists in determining (*a*) the existence of actual disease, and (*b*) its character, whether simple neurasthenia, severe hysteria, or an organic lesion. The outlook for ultimate recovery is good except in cases which present the more serious symptoms above mentioned. Nevertheless, it must be borne

in mind that traumatic hysteria is one of the most intractable affections which we are called upon to treat.

Treatment of Neurasthenia.—Many patients come under our care a generation too late for satisfactory treatment, and it may be impossible to restore the exhausted capital. In other instances, the recovery takes place rapidly, the patient remains well for a few months or a year, and then overwork, or even the ordinary wear and tear of life, again prostrates him. Other persons drift into a condition of chronic invalidism or become slaves to morphia or chloral. In the case of business or professional men, in whom the condition develops as a result of overwork or overstudy, it may be sufficient to enjoin absolute rest with change of scene and diet. A trip abroad, with a residence for a month or two in Switzerland, or, if there are symptoms of nervous dyspepsia, a residence at one of the Spas, will usually prove sufficient. The excitement of the large cities abroad should be avoided. Better still for these cases, if they carry it out, is a life in the woods or on the plains. Three months of tent-life in the Adirondacks or the same length of time in the Rocky Mountains will sometimes cure the most marked cases of this kind. Such a plan is not, however, within the circumstances of all. In a much larger class, including a large proportion of neurasthenic women, a systematic Weir Mitchell treatment rigidly carried out should be tried (see hysteria). For obstinate and protracted cases, particularly if combined with the chloral or morphia habit, no other plan is so satisfactory. The treatment of the gastric and intestinal symptoms so important in this condition has already been considered. In milder grades of the condition massage alone will be found very useful. For the irregular pains, particularly in the back and neck, the thermo-cautery is invaluable. Medicines are of little avail. Strychnia in full doses is often beneficial. For the relief of sleeplessness all possible measures should be resorted to before the employment of drugs.

XIV. OTHER FORMS OF FUNCTIONAL PARALYSIS.

I. Periodical Paralysis.

I have already referred to the remarkable periodical paralysis of the ocular muscles, which may recur at intervals for many years. There is a form of periodical paralysis involving the general muscles, which may recur with great regularity, and which is also a "family" affection. In Westphal's case, a boy of twelve, the attacks began in the eighth year, and at first recurred every four or six weeks, and lasted from a few hours to two days. Goldflam* has described a family in which twelve members were affected with this disease, the heredity being through the mother.

* Zeitschrift für klinische Medicin, Bd. xix, 1891.

Cousot has also met with a family in which the mother and four children were attacked. The disease occurs in youth, and the tendency to the attacks diminishes with age.

The clinical picture is very much alike in all the recorded cases. The paralysis involves, as a rule, the arms and legs. It comes on when the patients are in full health, and without any apparent cause, often during sleep. Sometimes it begins with weakness in the limbs, a sensation of weariness and sleepiness, not often with sensory symptoms. The paralysis is usually complete within the first twenty-four hours, beginning in the legs, to which in rare instances it is confined. The muscles of the neck are sometimes involved, and occasionally those of the tongue and pharynx. The cerebral nerves and the special senses are, as a rule, uninvolved. The attacks are afebrile, sometimes with low temperatures and slow pulse. The deep reflexes are reduced, sometimes abolished, and the skin reflexes may be feeble. One of the most remarkable features is the extraordinary reduction or complete abolition of the faradic excitability, both of muscles and of nerves.

Improvement begins sometimes in the course of a few hours or after a day or two, and the paralysis disappears completely, and the patient is perfectly well. As mentioned, the attacks may recur every few weeks, in some instances even daily; more commonly, an interval of one or two weeks elapses between the attacks. Goldflam suggests that the paralysis is due to an auto-intoxication, and that the poisonous material acts upon the nerve-endings in the muscles. He has made experiments with the urine of a case which showed that during the attacks the toxic properties of this secretion were materially increased. From the recurring, periodic character of the attacks they have been supposed to be due to malaria, but of this there is no evidence.

II. Astasia; Abasia.

These terms, indicating respectively inability to stand and inability to walk, have been applied by Charcot and Blocq to diseased conditions characterized by loss of the power of standing or of walking with retention of muscular power, coördination, and sensation. Blocq's definition is as follows: "A morbid state in which the impossibility of standing erect and walking normally is in contrast with the integrity of sensation, of muscular strength, and of the coördination of the other movements of the lower extremities." The condition forms a symptom group, not a morbid entity, and is probably a functional neurosis. Knapp in a recent paper analyzes the 50 cases reported in the literature. Twenty-five of these were in men, 25 in women. In 21 cases hysteria was present; in 3, chorea; in 2, epilepsy; and in 4, intention psychoses. As a rule, the patients, though able to move the feet and legs perfectly when in bed, are either unable to walk properly or cannot stand at all. The disturbances have been very varied,

and different forms have been recognized. The commonest, according to Knapp's analysis of the recorded cases, is the paralytic, in which the legs give out as the patient attempts to walk and "bend under him as if made of cotton." "There is no rigidity, no spasm, no incoördination. In bed, sitting, or even while suspended, the muscular strength is found to be good." Other cases are associated with spasm or ataxia; thus there may be movements which stiffen the legs and give to the gait a somewhat spastic character. In other instances there are sudden flexions of the legs, or even of the arms, or a saltatory, spring-like spasm. In a majority of the cases it is a manifestation of a neurosis allied to hysteria.

The cases, as a rule, recover, particularly in young persons. Relapses are not uncommon. The rest treatment and static electricity should be employed.

V. VASO-MOTOR AND TROPHIC DISORDERS.

I. RAYNAUD'S DISEASE.

Definition.—A vascular disorder, probably dependent upon vaso-motor influences, characterized by three grades of intensity: (*a*) Local syncope, (*b*) local asphyxia, and (*c*) local or symmetrical gangrene.

Local Syncope.—This condition is seen most frequently in the extremities, producing the condition known as dead fingers or dead toes. It is analogous to that produced by great cold. The entire hand may be affected with the fingers; more commonly only one or more of the fingers. This feature of the disease rarely occurs alone, but is generally associated with local asphyxia. The common sequence is as follows: On exposure to slight cold or in consequence of some emotional disturbance the fingers become white and cold, or both fingers and toes are affected. The pallor may continue for an indefinite time, though usually not more than an hour or so; then gradually a reaction follows and the fingers get burning hot and red. This does not necessarily occur in all the fingers together; one finger may be as white as marble, while the adjacent ones are of a deep red or plum color.

Local Asphyxia.—Chilblains form the mildest grade of this condition. It usually follows the local syncope, but it may come on independently. The fingers and toes are oftenest affected, next in order the ears; more rarely portions of the skin on the arms and legs. During an attack the fingers alone, sometimes the hands, also swell and become intensely congested. In the most extreme grade the fingers are perfectly livid, and the capillary circulation is almost stagnant. The swelling causes stiffness and usually pain, not acute, but due to the tension and distention of the skin. Sometimes there is marked anæsthesia. Attacks of this sort

may recur for years, and be brought on by the slightest exposure to cold or in consequence of disturbances, either mental or, in some instances, gastric. Apart from this unpleasant symptom the general health may be very good. The attacks may recur only at long intervals or during the winter time.

Local or Symmetrical Gangrene.—The mildest grade of this condition follows the local asphyxia, in the chronic cases of which small necrotic areas are sometimes seen at the tips of the fingers. Sometimes the pads of the fingers and of the toes are quite cicatricial from repeated slight losses of this kind. So also when the ears are affected there may be superficial loss of substance at the edge. The severer cases, which terminate in extensive gangrene, are fortunately rare.

In an attack the local asphyxia persists in the fingers. The terminal phalanges, or perhaps only one finger, become black, cold, and insensible. The skin begins to necrose and superficial gangrenous blebs appear. Gradually a line of demarkation shows itself and a portion of one or more of the fingers sloughs away. The resulting loss of substance is much less than the appearance of the hand or foot would indicate, and a condition which looks as if the patient would lose all the fingers or half of a foot may result perhaps in only a slight superficial loss in the phalanges. In severer cases the greater portion of a finger or the tip of the nose may be lost. Occasionally the disease is not confined to the extremities, but affects symmetrical patches on the limbs or trunk, and may pass on to rapid gangrene. These severe types of cases occur particularly in young children, and death may result within three or four days. The attacks are usually very painful, and the motion of the part is much impaired. In some cases numbness and tingling persist for a long time.

There are remarkable concomitant symptoms in Raynaud's disease to which a good deal of attention has been paid of late years. Hæmoglobinuria may develop during an attack, or may take the place of an outbreak. In such instances the affection is usually brought on by cold weather. In a case reported by H. M. Thomas from my clinic, Raynaud's disease occurred for three successive winters and always in association with hæmoglobinuria. The attacks were sometimes preceded by a chill. Several cases of the kind are found in Barlow's appendix to his translation of Raynaud's paper for the New Sydenham Society. The onset with a chill, as in the case just mentioned, has doubtless given rise to the idea that the disease is in some way associated with ague. Cerebral symptoms, particularly mental torpor and transient loss of consciousness, have also been noticed in some cases. The case just mentioned with hæmoglobinuria had epilepsy with the attacks. Exposure on a cold day would bring on an epileptic seizure with the local asphyxia and bloody urine. Occasionally joint affections develop, particularly anchylosis and thickening of the phalangeal articulations. Southey has reported a case in which mania developed, and Barlow an instance in which the woman had delusions. Peripheral neuritis has been found in several cases.

The *pathology* of this remarkable disease is still obscure. Raynaud suggested that the local syncope was produced by contraction of the vessels, which seems likely. The asphyxia is dependent upon dilatation of the capillaries and small veins, probably with the persistence of some degree of spasm of the smaller arteries. There are two totally different forms of congestion, which may be shown in adjacent fingers; one may be swollen, of a vivid red color, extremely hot, the capillaries and all the vessels fully distended, and the anæmia produced by pressure may be instantaneously obliterated; the adjacent finger may be equally swollen, absolutely cyanotic, stone cold, and the anæmia produced by pressure takes a long time to disappear. In the latter case the arterioles are probably still in a condition of spasm.

Treatment.—In many cases the attacks recur for years uninfluenced by treatment. Mild attacks require no treatment. In the severer forms of local asphyxia, if in the feet, the patient should be kept in bed with the legs elevated. The toes should be wrapped in cotton-wool. The pain is often very intense and may require morphia. Carefully applied, systematic massage of the extremities is sometimes of benefit. Galvanism may be tried. Barlow advises immersing the affected limb in salt water and placing one electrode over the spine and the other in the water.

II. ANGIO-NEUROTIC ŒDEMA.

Definition.—An affection characterized by the occurrence of local œdematous swellings, more or less limited in extent, and of transient duration. Severe colic is sometimes associated with the outbreak. There is a marked hereditary disposition in the disease. The affection has been specially studied by Quincke, Jamieson, J. E. Graham, and Matas.

Symptoms.—The œdema appears suddenly and is usually circumscribed. It may appear in the face; the eyelid is a common situation; or it may involve the lips or cheek. The backs of the hands, the legs, or the throat may be attacked. Usually the condition is transient, associated perhaps with slight gastro-intestinal distress, and the affection is of little moment. There may be a remarkable periodicity in the outbreak of the œdema. In Matas's case this periodicity was very striking; the attack came on every day at eleven or twelve o'clock. The disease may be hereditary through many generations. In the family whose history I reported, five generations had been affected, including twenty-two members. The swellings appear in various parts; only rarely are they constant in one locality. The hands, face, and genitalia are the parts most frequently affected. Itching, heat, redness, or, in some instances, urticaria may precede the outbreak. Sudden œdema of the larynx may prove fatal. Two members of the family just referred to died of this complication. In one member of this family, whom I saw repeatedly in attacks, the swell-

ings came on in different parts; for example, the under lip would be swollen to such a degree that the mouth could not be opened. The hands enlarge suddenly, so that the fingers cannot be bent. The attacks recur every three or four weeks. Accompanying them are usually gastro-intestinal attacks, severe colic, pain, nausea, and sometimes vomiting. The colic is of great intensity and usually requires morphia. Arthritis apparently does not occur.

The disease has affinities with urticaria, the giant form of which is probably the same disease. There is a form of severe purpura, often with urticarial manifestations, which is also associated with marked gastro-intestinal crises. Quincke regards the condition as a vaso-motor neurosis, under the influence of which the permeability of the vessels is suddenly increased.

The *treatment* is very unsatisfactory. In the cases associated with anæmia and general nervousness, tonics, particularly large doses of strychnia, do good; but too often the disease resists all treatment.

III. FACIAL HEMI-ATROPHY.

An affection characterized by progressive wasting of the bones and soft tissues of one side of the face. The atrophy begins, as a rule, in childhood, but in a few cases has not come on until middle age. It begins diffusely, but in some instances has started at one spot on the skin and has gradually spread, involving at first the subcutaneous tissues, then the muscles and the bones, more particularly the upper jaw. The wasting is sharply limited at the middle line, and the appearance of the patient is very remarkable, the face looking as if made up of two halves from different persons. There is usually change in the color of the skin and the hair falls. Owing to the wasting of the alveolar processes the teeth become loose and ultimately fall out. The wasting involves the tissues of the orbit, and the eye on the affected side is sunken. In a majority of the cases the atrophy has been confined to one side of the face, but there are instances on record in which the disease was bilateral, and a few cases in which there were areas of atrophy on the back and on the arm of the same side. The disease is rare. Sachs has collected 97 cases from the literature.

Two autopsies have been made. In Mendel's case there was the terminal stage of an interstitial neuritis in all the branches of the trigeminus, from its origin to the periphery, most marked in the superior maxillary branch.

In Homén's case, which came on rapidly and scarcely belongs to the typical form of the disease, a tumor was found pressing upon the Gasserian ganglion and the trigeminus nerve.

The disease is recognized at a glance. The facial asymmetry asso-

ciated with congenital wryneck must not be confounded with progressive facial hemi-atrophy. The precise nature of the disease is still doubtful.

IV. ACROMEGALIA.

Definition.—A dystrophy characterized by abnormal processes of growth, chiefly in the bones of the face and extremities.

The term was introduced by Marie, and signifies large extremities.

Etiology.—Nothing definite is known concerning the cause of the disease. It occurs rather more frequently in women. Of the 38 cases analyzed in the monograph of Souza-Leite, 16 were in men and 22 in women. The disease usually begins about the twenty-fifth year, though in some instances as late as the fortieth. Rheumatism, syphilis, and the specific fevers have preceded the development of the disease, but probably have no special connection with it. In this country five or six cases have been reported, two by J. E. Graham, of Toronto.

Symptoms.—In a well-marked case the disease presents most characteristic features. The hands and feet are greatly enlarged, but are not deformed, and can be used freely. The hypertrophy is general, involving all the tissues, and gives a curious spade-like character to the hands. The wrists may be enlarged, but the arms are rarely affected. The feet are involved like the hands and are uniformly enlarged. The big toe may be much larger in proportion. The nails are usually broad and large. The head increases in volume, but not as much in proportion as the face, which becomes much elongated and enlarged in consequence of the increase in the size of the superior and inferior maxillary bones. The latter in particular increases greatly in size, and often projects below the upper jaw. The alveolar processes are widened and the teeth separated. The soft parts also increase in size, and the nostrils are large and broad. The eyelids are sometimes greatly thickened, and the ears enormously hypertrophied. The tongue in some instances becomes greatly enlarged. Late in the disease the spine may be affected and the back bowed—kyphosis. The bones of the thorax may slowly and progressively enlarge. With this gradual increase in size the skin of the hands and face may appear normal. Sometimes it is slightly altered in color, coarse, or flabby, but it has not the dry, harsh appearance of the skin in myxœdema. The muscles are sometimes wasted. Changes in the thyroid have been found, but are not constant. The gland has been normal in some, atrophied in others, and in a third group of cases enlarged. Erb, who has made an elaborate study of the disease, has noticed an area of dulness over the manubrium sterni, which he thought possibly due to the persistence or enlargement of the thymus. Headache is not uncommon. Menstrual disturbance may occur early, and there may be suppression. In some instances vision has been involved, owing to a gradual atrophy of

the optic nerve. The disease may persist for fifteen, twenty, or more years.

The *pathological anatomy* has been studied in a few cases. In addition to enlargement of the bones, which is a true hypertrophy, enormous enlargement of the hypophysis (pituitary body) has been found, and some have regarded the disease as associated in some way with this. Less constant have been the changes in the thymus and in the thyroid. In some instances the peripheral nerves have been involved. The most exhaustive anatomical study made as yet is that published by Arnold, of Heidelberg, on the case which was described clinically by Friedreich and Erb.

As stated, the true nature of the disease is unknown. Marie regards it as a systemic dystrophy, analogous to myxœdema and possibly due to the morbid condition of the pituitary body, just as myxœdema is associated with disease of the thyroid.

Diagnosis.—The disease must be carefully separated from the *osteitis deformans* of Paget, in which the shafts of the long bones are chiefly involved, and in the head the bones of the cranium, but not those of the face. Marie states that in Paget's disease the face is triangular with the base upward; in acromegalia it is ovoid, or egg-shaped, with the large end downward; while in myxœdema it is round and full-moon shaped. The disease must not be confounded with the instances of congenital or progressive hypertrophy of a single member, as of the leg or arm, the so-called giant growth, in which the various proportions are maintained.

Lastly, Marie has separated from acromegalia a group of cases characterized by hypertrophy of the bones of the extremities and of the shafts, producing great disability. The spine is also affected and curvature takes place. The fingers are characteristic. The terminal phalanges become bulbous, enlarged, and the nails are curved, which gives the appearance of the so-called Hippocratic finger, a very different condition indeed from the flattened terminal phalanges of acromegalia. Etiologically, Marie regards this form as associated in some way with pulmonary troubles. Thus, for instance, two of the patients had purulent pleurisy, the cases of Ewald and of Saundby had new growths in the lungs, and others presented chronic bronchitis. Marie, therefore, terms this form *osteo-arthropathie pneumique*. It is doubtful, however, as Arnold states in his exhaustive study of Friedreich's case, whether this form can really be separated from acromegalia.

The treatment does not appear to have any influence upon the progress of the disease.

Here may be mentioned a remarkable dystrophy, met with so far only in women, known as *sclerodactyle*, in which there are symmetrical involvements of the fingers, which become deformed, shortened, and atrophied. The skin becomes thickened, of a waxy color, and is sometimes pigmented. Bullæ and ulcerations have been met with in some instances, and a great deformity of the nails. The disease has usually followed exposure, and the

patients are much worse during the winter and are curiously sensitive to cold. There may be changes in the skin of the feet, but the deformity similar to that which occurs in the hand has not been noted. Some of the cases have presented in addition diffuse sclerodermatous changes of the skin of other parts. An admirable description of the disease has been given by Gordinier.*

V. SCLERODERMA.

Definition.—A condition of localized or diffuse induration of the skin.

Two forms are recognized, the localized or circumscribed, which corresponds to the keloid of Addison and to morphea, and the diffuse, in which large areas are involved.

In the *circumscribed form* there are patches, ranging from a few centimetres in diameter to the size of the hand or larger, in which the skin has a waxy or dead-white appearance and to the touch is brawny, hard, and inelastic. Sometimes there is a preliminary hyperæmia of the skin, and subsequently there are changes in color, either areas of pigmentation or of complete atrophy of the pigment—leucoderma. The sensory changes are rarely marked. The secretion of sweat is diminished or entirely abolished. The disease is more common in women than in men, and is situated most frequently about the breasts and neck, sometimes in the course of the nerves. The patches may develop with great rapidity, and may persist for months or years; sometimes they disappear in a few weeks.

The *diffuse form*, though less common, is more serious. It develops first in the extremities or in the face, and the patient notices that the skin is unusually hard and firm, or that there is a sense of stiffness or tension in making accustomed movements. Gradually a diffuse, brawny induration develops and the skin becomes firm and hard, and so united to the subcutaneous tissues that it cannot be picked up or pinched. The skin may look natural, but more commonly is glossy, drier than normal, and unusually smooth. Of 44 cases, in 24 the first appearances were on the arms, in 7 on the legs, in 1 on both, in 10 on the face and neck, and in 2 on the trunk (Dinkler). The disease may gradually extend and involve the skin of an entire limb; in rare cases, it becomes universal, the face is expressionless, the lips cannot be moved, mastication is impossible, and it becomes extremely difficult to feed the patient. The hands become fixed, the fingers immobile, on account of the extreme induration of the skin over the joints. The disease is chronic, lasting for many months or many years. There are instances on record of its persistence for more than twenty years. Recovery may occur, or the disease may be arrested.

* American Journal of the Medical Sciences, January, 1889.

The patients are apt to succumb to pulmonary complaints or to nephritis. Rheumatic troubles have been noticed in some instances; in others, endocarditis. The pathology of the disease is unknown. It is usually regarded as a tropho-neurosis, probably dependent upon changes in the arteries of the skin leading to connective-tissue overgrowth.

The patients require to be warmly clad and to be guarded against exposure, as they are particularly sensitive to changes in the weather. Frictions with oil, and galvanism are recommended.

AINHUM.

Here a brief reference may be made to the remarkable trophic lesion described by Da Silva Lima, which is met with in negroes in Brazil, Africa, India, and occasionally in the Southern States. It is confined to the toes, usually the little toe, and begins as a furrow on the line of the digito-plantar fold. This gradually deepens, the end of the toe enlarges, and, usually without inflammation or pain, the toe falls off. The process may last some years. Cases have been reported in this country by Hornaday, Pittman, F. J. Shepherd, and Morrison.

SECTION IX.

DISEASES OF THE MUSCLES.

I. MYOSITIS.

Definition.—Inflammation of the voluntary muscles.

A primary myositis occurs as an acute or subacute affection, and is probably dependent on some unknown infectious agent. Several characteristic cases have been described of late years. The case of E. Wagner may be taken as a typical example. A tuberculous but well-built woman entered the hospital, complaining of stiffness in the shoulders and a slight œdema of the back of the hands and forearms. There was paræsthesia, the arms became swollen, the skin tense, and the muscles felt doughy. Gradually the thighs became affected. The disease lasted about three months. The post-mortem showed slight pulmonary tuberculosis; all the muscles except the glutei, the calf, and abdominal muscles were stiff and firm, but fragile, and there were serous infiltration, great proliferation of the interstitial tissue, and fatty degeneration. Similar cases have been reported by Unverricht, Hepp, and Jacoby of New York. In the case reported by Jacoby the muscles were firm, hard, and tender, and there was slight œdema of the skin The duration of the cases is usually from one to three months, though there are instances in which it has been longer The swelling and tenderness of the muscles, the œdema, and the pain naturally suggest trichinosis, and indeed Hepp speaks of it as a pseudo-trichinosis. The nature of the disease is unknown. Senator's case presented marked disorders of sensation, and there is a question whether the peripheral nerves are not involved with the muscles. Wagner suggests that some of these cases were examples of acute progressive muscular atrophy. The separation from trichinosis can be made only by removing a portion of the muscle. There are septic cases in which a diffuse, purulent infiltration of the muscles of different regions occurs. Instances have been reported in which this has been described as the primary affection, the condition of the muscles even passing on to gangrene.

A remarkable affection is *myositis ossificans progressiva*, in which portions of the muscles undergo a progressive calcification.

II. IDIOPATHIC MUSCULAR ATROPHY

(Primary Muscular Dystrophy—Erb).

Definition.—Muscular wasting, with or without an initial hypertrophy, beginning in various groups of muscles, usually progressive in character, and dependent on primary changes in the muscles themselves. A marked hereditary disposition is met with in the disease.

Before considering the primary muscular atrophies it may be well to summarize briefly the chief conditions under which muscular atrophy occurs. These are:

(1) Acute or chronic lesions of the nuclei of the motor path, which may be (*a*) cortical, as a direct result of a cerebral lesion; (*b*) medullary, as in chronic bulbar paralysis; (*c*) spinal, either acute, as in poliomyelitis of children, or chronic, as in the progressive muscular atrophy of the simple or of the spastic type.

(2) Neuritic muscular atrophy, following a local neuritis due to trauma, a multiple neuritis due to alcohol, lead, and the infectious diseases. In this same category probably may be placed the muscular atrophies associated with joint-disease, the progressive hemi-atrophy of the face, and the atrophy sometimes found in cases of hysteria.

(3) Conditions of the muscles themselves—primary muscular atrophy.

Etiology.—The most important factor is heredity. Many members of the same family may be attacked through several generations. Males, as a rule, are more frequently affected than females. The disease is usually transmitted through the mother, though she may not herself be the subject. As many as twenty or thirty cases have been described in five generations. Isolated cases, however, are not uncommon. The disease usually sets in before puberty, but may be as late as the twentieth or twenty-fifth year, or in some instances even later. No etiological factors of any moment are known other than heredity.

Clinical Forms.—Two chief types may be recognized: (1) With primary hypertrophy, the pseudo-hypertrophic muscular paralysis; and (2) with primary atrophy.

Pseudo-hypertrophic Muscular Paralysis.—The first symptom noticed is, as a rule, clumsiness in the movements of the child, and on examination certain muscles or groups of muscles seem to be enlarged, particularly those of the calves. The extensors of the leg, the glutei, the lumbar muscles, the deltoid, triceps, and infraspinatus, are the next most frequently involved, and may stand out with great prominence. The muscles of the neck, face, and forearm rarely suffer. Sometimes only a portion of a muscle is involved. With this hypertrophy of some muscles there is wasting of others, particularly the lower portion of the pectorals and the latissimus dorsi. The attitude when standing is very characteristic. The legs are far apart, the shoulders thrown back, the spine is greatly curved, and the abdomen protrudes. The gait is waddling and awkward. In

getting up from the floor the position assumed, as so well known now through Gowers's figures, is pathognomonic. The patient first turns over in the all-fours position and raises the trunk with his arms; the hands are then moved along the ground until the knees are reached; then with one hand upon a knee he lifts himself up, grasps the other knee, and gradually pushes himself into the erect posture, as it has been expressed, by climbing up his legs. The striking contrast between the feebleness of the child and the powerful-looking pseudo-hypertrophic muscles is very characteristic.

The course of the disease is slow, but progressive. Wasting proceeds and finally all traces of the enlarged condition of the muscles disappears. At this late period distortions and contractions are common.

Primary Atrophic Form.—Here, too, there is the same marked tendency to involvement of different members of a family. Five or six different types have been described, but it seems more rational to group them together under the designation of idiopathic muscular atrophy. In all of the cases the atrophy begins, as a rule, before the twentieth year. According to the site of the primary atrophy different forms have been described. In the *juvenile type* of Erb the affection begins about the fifteenth or the twentieth year and involves the muscles of the upper arm and shoulder and the gluteal and thigh muscles. In the facio-scapulo-humeral type of Landouzy and Déjérine the muscles of the face are early involved with those of scapulo-humeral groups. This form occurs usually in families, and the onset may be delayed until the twentieth or thirtieth year. Leyden describes an *hereditary form*, beginning in the lower extremities and back, which may be associated with hypertrophy of the calves. Another type has been described by Charcot and Tooth—the *peroneal form;* but there is still some doubt whether this is not in reality a myelopathy and more closely related to chronic polio-myelitis anterior. In this form the atrophy begins in the muscles of the legs, usually in the extensors of the great toe, and afterward in the common extensors and the peroneal groups. The cases usually begin early, and the heredity through the mother has been traced in several remarkable series, particularly that of Herringham's. Fibrillary contractions and the reaction of degeneration are present. Nerve degeneration has been found in the peripheral parts, and ascending degeneration of the columns of Goll.

Morbid Anatomy.—The spinal cord and peripheral nerves have been found normal in cases of pseudo-hypertrophic muscular paralysis and in the forms of idiopathic muscular atrophy. The muscles in the pseudo-hypertrophic condition present great variations in the size of the muscle fibres, some of which may be hypertrophied and others wasted. In the early stage the hypertrophy of the fibres may be very pronounced and the nuclei of the sarcolemma are greatly increased. In some instances, too, the fibres have been fissured longitudinally. At a later stage the muscular

fibres are wasted and largely replaced by connective tissue and fat. In the primary atrophic form wasting of the fibres, increase in the interstitial tissue, and the development of fat are the most marked features. Except in the peroneal type, about which there is still doubt, no affection of the nerves or cord has been determined.

Diagnosis.—The primary myopathies can usually be readily distinguished from the cerebral, myelopathic, and neuritic forms.

(*a*) In the cerebral atrophy loss of power usually precedes the atrophy, which is either of a monoplegic or hemiplegic type.

(*b*) In the myelopathic or spinal muscular atrophy the distinctions are clearly marked. *Polio-myelitis anterior chronica* begins in the small muscles of the hand, a situation rarely if ever affected by the primary myopathies, which involve first those of the calves, the trunk, the face, or the shoulder-girdle. In the myelopathic atrophy the reaction of degeneration is present and fibrillary twitchings occur in both the atrophied and non-atrophied muscles. In many cases in addition to the wasting in the arms there is a spastic condition in the legs and increase in the reflexes. The myelopathic atrophies come on late in life; the myopathic forms develop, as a rule, early. In the primary muscular atrophies heredity plays an important *rôle*, which in the myelopathic is quite subsidiary.

(*c*) In the neuritic muscular atrophies, whether due to lead or to trauma, the general characters and the mode of onset are distinctive. In the cases of multiple neuritis seen for the first time at a period when the wasting is marked there is often difficulty, but the absence of family history and the distribution are important features. Moreover, the paralysis is out of proportion to the atrophy. Sensory symptoms may be present, and in the cases in which the legs are chiefly involved there is usually the *steppage* gait so characteristic of peripheral neuritis.

The outlook in the primary myopathies is bad. The wasting progresses uniformly, uninfluenced by treatment. Erb holds that by electricity and massage the progress is occasionally arrested. The general health should be carefully looked after, moderate exercise allowed, frictions with oil applied to the muscles, and when the patient becomes bedfast, as is inevitable sooner or later, care should be taken to prevent contractures in awkward positions.

III. THOMSEN'S DISEASE; MYOTONIA CONGENITA.

Definition.—An hereditary disease characterized by tonic cramp of the muscles on attempting voluntary movements. The disease received its name from the physician who first described it, in whose family it has existed for five generations.

Etiology.—All the typical cases have occurred in family groups; a few isolated instances have been described in which similar symptoms

have been present. The disease is rare in this country and in England; it seems more common in Germany and in Scandinavia.

Symptoms.—The disease comes on in childhood. It is noticed that on account of the stiffness the children are not able to take part in ordinary games. The peculiarity is noticed only during voluntary movements. The contraction which the patient wills is slowly accomplished; the relaxation which the patient wills is also slow. The contraction often persists for a little time after he has dropped an object which he has picked up. In walking, the start is difficult; one leg is put forward slowly, it halts from stiffness for a second or two, and then after a few steps the legs become limber and he walks without any difficulty. The muscles of the arms and legs are those usually implicated; rarely facial, ocular, or laryngeal muscles. Emotion and cold aggravate the condition. In some instances there is mental weakness. The sensation and the reflexes are normal. The condition of the muscles is interesting. The patients appear and are muscular, and there is sometimes a definite hypertrophy of the muscles. The force is scarcely proportionate to the size. Erb has described a characteristic reaction of the nerve and muscle to the electrical currents—the so-called myotonic reaction, the chief feature of which is that normally the contractions caused by either current attain their maximum slowly and relax slowly, and vermicular, wave-like contractions pass from the cathode to the anode.

The disease is incurable, but it may be arrested temporarily. The nature of the affection is unknown. There is an extraordinary increase in the size of the voluntary fibres. According to Hale White,* who has recently treated the subject in an exhaustive and critical manner, the fibres may be more than double the width of those of the normal muscles. The nuclei and the interstitial tissue may be increased and some of the fibres contain vacuoles. No post-mortem has been made. No treatment for the condition is known.

IV. PARAMYOCLONUS MULTIPLEX.

An affection, described by Friedrich, characterized by clonic contractions, chiefly of the muscles of the extremities, occurring either constantly or in paroxysms.

The cases have usually been in males and the disease has followed emotional disturbance, fright, or straining. The contractions are usually bilateral and may vary from fifty to one hundred and fifty in the minute. Occasionally tonic spasms occur. It is not accompanied by any sensory or motor disturbances. In the intervals between the attacks there may be tremors of the muscles. In the severe spasms the movements may be very

* Guy's Hospital Reports, 1889.

violent; the body is tossed about, and it is sometimes difficult to keep the patient in bed. In a case which I saw at the Bicêtre the patient was perfectly quiet so long as his legs were tied down with a sheet, but as soon as this was removed the clonic spasms occurred in the legs and muscles of the back and tossed the body about in the bed from side to side. The patient uttered a curious expiratory grunt. The nature of the disease is unknown.

SECTION X.

THE INTOXICATIONS, SUN-STROKE, OBESITY.

I. ALCOHOLISM.

(1) **Acute Alcoholism.**—When a large quantity of alcohol is taken, its influence on the nervous system is manifested in muscular incoördination, mental disturbance, and, finally, narcosis. The individual presents a flushed, sometimes slightly cyanosed face, a full pulse, with deep but rarely stertorous respirations. The pupils are dilated. The temperature is frequently below normal, particularly if the patient has been exposed to cold. Perhaps the lowest reported temperatures have been in cases of this sort. An instance is on record in which the patient on admission to hospital had a temperature of 24° C. (ca. 75° F.), and ten hours later the temperature had not risen to 91°. The unconsciousness is rarely so deep that the patient cannot be roused to some extent, and in reply to questions he mutters incoherently. Muscular twitchings may occur, but rarely convulsions. The breath has a heavy alcoholic odor.

The diagnosis is not difficult, yet mistakes are frequently made. Persons are sometimes brought to hospital by the police supposed to be drunk when in reality they are dying from apoplexy. Too great care cannot be exercised, and the patient should receive the benefit of the doubt. In some instances the mistake has arisen from the fact that a person who has been drinking heavily has been stricken with apoplexy. In this condition the coma is usually deeper, stertor is present, and there may be evidence of hemiplegia in the greater flaccidity of the limbs on one side. The subject has already been considered in the section upon uræmic coma.

(2) **Chronic Alcoholism.**—In moderation, wine, beer, and spirits may be taken throughout a long life without impairing the general health.

According to Payne, the poisonous effects of alcohol are manifested (1) as a functional poison, as in acute narcosis; (2) as a tissue poison, in which its effects are seen on the parenchymatous elements, particularly epithelium and nerve, producing a slow degeneration, and on the blood-vessels, causing thickening and ultimately fibroid changes; and (3) as a checker

of tissue oxidation, since the alcohol is consumed in place of the fat. This leads to fatty changes and sometimes to a condition of general steatosis.

The chief effects of chronic alcohol poisoning may be thus summarized:

Nervous System.—Functional disturbance is common.—Unsteadiness of the muscles in performing any action is a constant feature. The tremor is best seen in the hands and in the tongue. The mental processes may be dull, particularly in the early morning hours, and the patient is unable to transact any business until he has had his accustomed stimulant. Irritability of temper, forgetfulness, and a change in the moral character of the individual gradually come on. The judgment is seriously impaired, the will enfeebled, and in the final stages dementia may supervene. The relation of chronic alcoholism to insanity has been much discussed. According to Savage, of 4,000 patients admitted to the Bethlehem Hospital, 133 gave drink as the cause of their insanity. Chronic alcoholism is believed by many to be one of the special causes of dementia paralytica, but the opinions of experts on this question are still discordant. Savage states that not more than seven per cent are caused by alcohol alone. In many cases it is certainly one of the important elements in the strain which leads to this breakdown.

No characteristic changes are found in the nervous system. Hæmorrhagic pachymeningitis is not very uncommon. Opacity and thickening of the pia-arachnoid membranes, with more or less wasting of the convolutions, generally occur. These are in no way peculiar to chronic alcoholism, but are found in old persons and in chronic wasting diseases. In the very protracted cases there may be chronic encephalo-meningitis with adhesions of the membranes. By far the most striking effect of alcohol on the nervous system is the production of the alcoholic neuritis, which has already been considered.

Digestive System.—Catarrh of the stomach is the most common symptom. The toper has a furred tongue, heavy breath, and in the morning a sensation of sinking at the stomach until he has his dram. The appetite is usually impaired and the bowels are constipated. These features are associated with a chronic catarrh of the stomach.

Alcohol produces definite changes on the liver, leading to the various forms of cirrhosis already described. The effect is probably a primary degenerative change in the liver-cells, although many good observers still hold that the poison acts first upon the connective-tissue elements. It is probable that a special vulnerability of the liver-cells is necessary in the etiology of alcoholic cirrhosis. There are cases in which comparatively moderate drinking for a few years has been followed by cirrhosis; on the other hand, the livers of persons who have been steady drinkers for thirty or forty years may show only a moderate grade of sclerosis. With the gastric and hepatic disorders the facies often becomes very characteristic. The venules of the cheeks and nose are dilated; the latter becomes enlarged,

red, and may present the condition known as *acne rosacea*. The eyes are watery, the conjunctivæ hyperæmic and sometimes bile-tinged.

Kidneys.—The influence of chronic alcoholism upon these organs is by no means so marked. According to Dickinson the total of renal disease is not greater in the drinking class, and he holds that the effect of alcohol on the kidneys has been much overrated. Formad has directed attention to the fact that in a large proportion of chronic alcoholics the kidneys are increased in size. The Guy's Hospital statistics support this statement, and Pitt notes that in forty-three per cent of the bodies of hard drinkers the kidneys were hypertrophied without showing morbid change. The typical granular kidney seems to result indirectly from alcohol through the arterial changes.

It was formerly thought that alcohol was in some way antagonistic to tuberculous disease, but the observations of late years indicate clearly that the reverse is the case and that chronic drinkers are much more liable to both acute and pulmonary tuberculosis. It is probably altogether a question of altered tissue-soil, the alcohol lowering the vitality and enabling the bacilli more readily to develop and grow.

(3) **Delirium Tremens** (*mania a potu*) is really only an incident in the history of chronic alcoholism, and results from the long-continued action of the poison on the brain. The condition was first accurately described early in this century by Sutton, of Greenwich, who had numerous opportunities for studying the different forms among the sailors. One of the most thorough and careful studies of the disease was made by Ware, of Boston. A spree in a temperate person, no matter how prolonged, is rarely if ever followed by delirium tremens; but in the case of an habitual drinker a temporary excess is apt to bring on an attack. It sometimes develops in consequence of the sudden withdrawal of the alcohol. There are circumstances which in a heavy drinker determine, sometimes with abruptness, the onset of delirium. Such are an accident, a sudden fright or shock, and an acute inflammation, particularly pneumonia. At the outset of the attack the patient is restless and depressed and sleeps badly, symptoms which cause him to take alcohol more freely. After a day or two the characteristic delirium sets in. The patient talks constantly and incoherently; he is incessantly in motion, and desires to go out and attend to some imaginary business. Hallucinations of sight and hearing develop. He sees objects in the room, such as rats, mice, or snakes, and fancies that they are crawling over his body. The terror inspired by these imaginary objects is great, and has given the popular name "horrors" to the disease. The patients need to be watched constantly, for in their delusions they may jump out of the window or escape. Auditory hallucinations are not so common, but the patient may complain of hearing the roar of animals or the threats of imaginary enemies. There is much muscular tremor; the tongue is covered with a thick white fur, and when protruded is tremulous. The pulse is soft, rapid, and readily compressed. There is usually

fever, but the temperature rarely registers above 102° or 103°. In fatal cases it may be higher. Insomnia is a constant feature. On the third or fourth day in favorable cases the restlessness abates, the patient sleeps, and improvement gradually sets in. The tremor persists for some days, the hallucinations gradually disappear, and the appetite returns. In more serious cases the insomnia persists, the delirium is incessant, the pulse becomes more frequent and feeble, the tongue dry, the prostration extreme, and death takes place from gradual heart-failure.

Diagnosis.—The clinical picture of the disease can scarcely be confounded with any other. Cases with fever, however, may be mistaken for meningitis. By far the most common error is to overlook some local disease, such as pneumonia or erysipelas, or an accident, as a fractured rib, which in a chronic drinker may precipitate an attack of delirium tremens. In every instance a careful examination should be made, particularly of the lungs. It is to be remembered that in the severer forms, particularly the febrile cases, congestion of the bases of the lungs is by no means uncommon. Another point to be borne in mind is the fact that pneumonia of the apex is apt to be accompanied by delirium similar to *mania a potu*.

Prognosis.—Recovery takes place in a large proportion of the cases in private practice. In hospital practice, particularly in the large city hospitals to which the debilitated patients are taken, the death rate is higher. Gerhard states that of 1,241 cases admitted to the Philadelphia Hospital 121 proved fatal. Recurrence is frequent, almost indeed the rule, if the drinking is kept up.

Treatment.—Acute alcoholism rarely requires any special measures, as the patient sleeps off the effects of the debauch. In the case of profound alcoholic coma it may be advisable to wash out the stomach, and if collapse symptoms occur the limbs should be rubbed and hot applications made to the body. Should convulsions supervene, chloroform may be carefully administered. In the acute, violent alcoholic mania the hypodermic injection of apomorphia, one eighth or one sixth of a grain, is usually very effectual, causing nausea and vomiting, and rapid disappearance of the maniacal symptoms.

Chronic alcoholism is a condition very difficult to treat, and once fully established the habit is rarely abandoned. The most obstinate cases are are those with marked hereditary tendency. Withdrawal of the alcohol is the first essential. This is most effectually accomplished by placing the patient in an institution, in which he can be carefully watched during the trying period of the first week or ten days of abstention. The absence of temptation in institution life is of special advantage. For the sleeplessness the bromides or hyoscine may be employed. Quinine and strychnine in tonic doses may be given. Cocaine or the fluid extract of coca has been recommended as a substitute for alcohol, but it is not of much service. Prolonged seclusion in a suitable institution is in reality the only

effectual means of cure. When the hereditary tendency is strongly developed a lapse into the drinking habits is almost inevitable.

In delirium tremens the patient should be confined to bed and carefully watched night and day. The danger of escape in these cases is very great, as the patient imagines himself pursued by enemies or demons. Flint mentions the case of a man who escaped in his night-clothes and ran barefooted for fifteen miles on the frozen ground before he was overtaken. The patient should not be strapped in bed, as this aggravates the delirium; sometimes, however, it may be necessary, in which case a sheet tied across the bed may be sufficient, and this is certainly better than violent restraint by three or four men. Alcohol should be withdrawn at once unless the pulse is feeble.

Delirium tremens is a disease which, in a large majority of cases, runs a course very slightly influenced by medicine. The indications for treatment are to procure sleep and to support the strength. In mild cases half a drachm of bromide of potassium combined with tincture of capsicum may be given every three hours. Chloral is often of great service, and may be given without hesitation unless the heart's action is feeble. Good results sometimes follow the hypodermic use of hyoscine, one one-hundredth of a grain. Opium must be used cautiously. A special merit of Ware's work was the demonstration that on a rational or expectant plan of treatment the percentage of recovery was greater than with the indiscriminate use of sedatives, which had been in vogue for many years. When opium is indicated it should be given as morphia, hypodermically. The effect should be carefully watched, and if after three or four quarter-grain doses have been given the patient is still restless and excited, it is best not to push it farther. When fever is present the tranquillizing effects of a cold douche or cold bath may be tried, or the cold pack. The large doses of digitalis formerly employed are not advisable.

Careful feeding is the most important element in the treatment of these cases. Milk and concentrated broths should be given at stated intervals. If the pulse becomes rapid and shows signs of flagging alcohol may be given in combination with the aromatic spirits of ammonia.

II. MORPHIA HABIT (*Morphiomania*; *Morphinism*).

This habit arises from the constant use of morphia—taken at first, as a rule, for the purpose of allaying pain. The craving is gradually engendered, and the habit in this way acquired. The injurious effects vary very much, and in the East, where opium-smoking is as common as tobacco-smoking with us, the ill effects are, according to good observers, not so striking.

The habit is particularly prevalent among women and physicians who use the hypodermic syringe for the alleviation of pain, as in neuralgia or

sciatica. The acquisition of the habit as a pure luxury is rare in this country.

The symptoms at first are slight, and moderate doses may be taken for months without serious injury and without disturbance of health. There are exceptional instances in which for a period of years excessive doses have been taken without deterioration of the mental or bodily functions. As a rule, the dose necessary to obtain the desired sensations has gradually to be increased. As the effects wear off the victim experiences sensations of lassitude and mental depression, accompanied often with slight nausea and epigastric distress, symptoms which are relieved by another dose of the drug. The confirmed opium-eater presents a very characteristic appearance. There is a sallowness of the complexion which is almost pathognomonic, and he becomes emaciated, gray, and prematurely aged. He is restless, irritable, and unable to remain quiet for any time. Itching is a common symptom. The sleep is disturbed, the appetite and digestion are deranged, and except when directly under the influence of the drug the mental condition is one of depression. Occasionally there are profuse sweats, which may be preceded by chills. The pupils, except when under the direct influence of the drug, are dilated, sometimes unequal. Persons addicted to morphia are inveterate liars, and no reliance whatever can be placed upon their statements. In many instances this is not confined to matters relating to the vice. In women the symptoms may be associated with those of pronounced hysteria or neurasthenia. The practice may be continued for an indefinite time, usually requiring increase in the dose until ultimately enormous quantities may be needed to obtain the desired effect. Finally a condition of asthenia is induced, in which the victim takes little or no food and dies from the extreme bodily debility.

The *treatment* of the morphia habit is extremely difficult, and can rarely be successfully carried out by the general practitioner. Isolation, systematic feeding, and gradual withdrawal of the drug are the essential elements. As a rule, the patients must be under control in an institution and should be in bed for the first ten days. It is best in a majority of cases to reduce the morphia gradually. The diet should consist of beef-juices, milk, and egg-white, which should be given at short intervals. The sufferings of the patients are usually very great, more particularly the abdominal pains, sometimes nausea and vomiting, and the distressing restlessness. Usually within a week or ten days the opium may be entirely withdrawn. In all cases the pulse should be carefully watched and, if feeble, stimulants should be given, with the aromatic spirits of ammonia and digitalis. For the extreme restlessness a hot bath is serviceable. The sleeplessness is the most distressing symptom, and various drugs may have to be resorted to, particularly hyoscine and sulphonal and sometimes, if the insomnia persists, morphia itself.

It is essential in the treatment of a case to be certain that the patient

has no means of obtaining morphia. Even under the favorable circumstances of seclusion in an institution, and constant watching by a night and a day nurse, I have known a patient to practice deception for a period of three months. After an apparent cure the patients are only too apt to lapse into the habit.

The condition is one which has become so common, and is so much on the increase, that physicians should exercise the utmost caution in prescribing morphia, particularly to female patients. Under no circumstances whatever should a patient with neuralgia or sciatica be allowed to use the hypodermic syringe, and it is even safer not to intrust this dangerous instrument to the hands of the nurse.

III. LEAD-POISONING (*Plumbism; Saturnism*).

Etiology.—The disease is wide-spread, particularly in lead-workers and among plumbers, painters, and glaziers. The metal is introduced into the system in many forms. Miners usually escape, but those engaged in the smelting of lead-ores are often attacked. Animals in the neighborhood of smelting furnaces have suffered with the disease, and even the birds that feed on the berries in the neighborhood may be affected. Men engaged in the white-lead factories are particularly prone to plumbism. Accidental contamination may come in many ways; most commonly by drinking water which has passed through lead pipes or been stored in lead-lined cisterns. Wines and cider which contain acids quickly become contaminated in contact with lead. It was the frequency of colic in certain of the cider districts of Devonshire which gave the name Devonshire colic, as the frequency of it in Poitou gave the name *colica Pictonum*. Among the innumerable sources of accidental contamination may be mentioned milk, various sorts of beverages, hair dyes, false teeth, and thread. A serious outbreak of lead-poisoning, which was investigated by David D. Stewart, occurred recently in Philadelphia, owing to the disgraceful adulteration of a baking-powder with chromate of lead, which was used to give a yellow tint to the cakes. Lead given medicinally rarely produces poisoning.

All ages are attacked, but J. J. Putnam states that children are relatively less liable. The largest number of cases occur between thirty and forty. According to Oliver, from whose recent Gulstonian lectures I here quote, females are more susceptible than males. He states that they are much more quickly brought under its influence, and in a recent epidemic in which a thousand cases were involved the proportion of females to males was four to one.

The lead gains entrance to the system through the lungs, the digestive organs, or the skin. Poisoning may follow the use of cosmetics containing lead. Through the lungs it is freely absorbed. The chief channel,

according to Oliver, is the digestive system. It is rapidly eliminated by the kidneys and skin, and is present in the urine of lead-workers. The susceptibility is remarkably varied. The symptoms may be manifest with a month of exposure. On the other hand, Tanquerel (des Planches) met with a case in a man who had been a lead-worker for fifty-two years.

Morbid Anatomy.—Small quantities of lead occur in the body in health. J. J. Putnam's reports show that of 150 persons not presenting symptoms of lead-poisoning traces of lead occurred in the urine of 25 per cent.

In chronic poisoning lead is found in the various organs. The affected muscles are yellow, fatty, and fibroid. The nerves present the features of a peripheral degenerative neuritis. The cord and the nerve-roots are, as a rule, uninvolved. In the primary atrophic form the ganglion cells of the anterior horns are probably involved. In the acute fatal cases there may be the most intense entero-colitis.

Clinical Forms.—*Acute Poisoning.*—We do not refer here to the accidental or suicidal cases, which present vomiting, pain in the abdomen, and collapse symptoms. In workers in lead there are several manifestations which follow a short time after exposure and set in acutely. There may be, in the first place, a rapidly developing anæmia. Acute neuritis has been described, and convulsions, epilepsy, and a delirium, which may be, as Stephen Mackenzie has noted, not unlike that produced by alcohol. There are also cases in which the gastro-intestinal symptoms are most intense and rapidly prove fatal. There was admitted under my care in the Philadelphia Hospital a painter, aged fifty, suffering with anæmia and severe abdominal pain, which had lasted about a week. He had vomiting, constipation at first, afterward severe diarrhœa and melæna, with distention and tenderness of the abdomen. There were albumen and tube-casts in the urine. The temperature was usually subnormal. Death occurred at the end of the second week. There was found the most intense entero-colitis with hæmorrhages and exudation. These acute forms develop more frequently in persons recently exposed, and, according to Mackenzie, are more frequent in winter than in summer.

Chronic poisoning presents the following symptoms:

(*a*) *Anæmia*, the so-called saturnine cachexia, which may be profound. As a rule, however, the corpuscles do not sink below 50 per cent.

(*b*) *Blue line* on gums, which is a valuable indication, but not invariably present. Two lines must be distinguished: one, at the margin between the gums and teeth, is on, not in the gums, and is readily removed by rinsing the mouth and cleansing the teeth. The other is the well-known characteristic blue-black line at the margin of the gum. The color is not uniform, but being in the papillæ of the gums the line is, as seen with a magnifying-glass, interrupted. The lead is absorbed and converted in the tissues into a black sulphide by the action of sulphuretted hydrogen from the tartar of the teeth. The line may form rapidly after exposure and

disappear within a few weeks, or may persist for many months. Philipson has noted the occurrence of a black line in miners, due to the deposition of carbon.

The most important symptoms of chronic lead-poisoning are colic, lead-palsy, and the encephalopathy. Of these, the colic is the most frequent. Of Tanquerel's cases, there were 1,217 of colic, 101 of paralysis, and 72 of encephalopathy.

(*c*) *Colic* is the most common symptom of chronic lead-poisoning. It is often preceded by gastric or intestinal symptoms, particularly constipation. The pain is over the whole abdomen. The colic is usually paroxysmal, like true colic, and is relieved by pressure. There is often, in addition, between the paroxysms a dull, heavy pain. There may be vomiting. During the attack, as Riegel noted, the pulse is increased in tension and the heart's action is retarded. The pupils are usually unequal (Oliver).

(*d*) *Lead-palsy.*—This is rarely a primary manifestation. The onset may be acute, subacute, or chronic. It usually develops without fever. In its distribution it may be partial, limited to a muscle or to certain muscle groups, or generalized, involving in a short time the muscles of the extremities and the trunk. Madame Déjérine-Klumpke recognizes the following *localized forms:*

(1) Anti-brachial type, paralysis of the extensors of the fingers and of the wrist. In this the musculo-spiral nerve is involved, causing the characteristic wrist-drop. The supinator longus usually escapes.

(2) Brachial type, which involves the deltoid, the biceps, the brachialis anticus, and the supinator longus, rarely the pectorals. The atrophy is of the scapulo-humeral form. It is bilateral, and sometimes follows the first form, but it may be primary.

(3) The Aran-Duchenne type, in which the small muscles of the hand and of the thenar and hypothenar eminences are involved. It produces a paralysis closely resembling that of the early stage of *polio-myelitis anterior chronica.* The atrophy is marked, and may be the first manifestation of the lead-palsy. Möbius has shown that this form is particularly developed in tailors.

(4) The peroneal type. According to Tanquerel, the lower limbs are involved in the proportion of thirteen to one hundred of the upper limbs. The lateral peroneal muscles, the extensor communis of the toes, and the extensor proprius of the big toe are involved, producing the *steppage* gait.

(5) Laryngeal form. Adductor paralysis has been noted by Morell Mackenzie and others in lead-palsy.

Generalized Palsies.—There may be a slow, chronic paralysis, gradually involving the extremities, beginning with the classical picture of wrist-drop. More frequently there is a rapid generalization, producing complete paralysis in all the muscles of the parts in a few days. It may pursue a course like an ascending paralysis, associated with rapid wasting

of all four limbs. Such cases, however, are very rare. Death has occurred by involvement of the diaphragm. Oliver reports a case of Philipson's in which complete paralysis supervened. Déjérine-Klumpke also recognizes a febrile form of general paralysis in lead-poisoning, which may closely resemble the subacute spinal paralysis of Duchenne.

There is also a primary saturnine muscular atrophy in which the weakness and wasting come on together and develop proportionately. It is this form, according to Gowers, which most frequently assumes the Aran-Duchenne type.

The electrical reactions are those of lesions of the lower motor segment, and have been described under lesions of the nerves. The degenerative reaction in its different grades may be present, depending upon the severity of the disease.

Usually with the onset of the paralysis there are pains in the legs and joints, the so-called saturnine arthralgias. As a rule, however, sensation is unaffected and the sensory nerves are not involved.

(*e*) The *cerebral symptoms* are numerous. Optic neuritis or neuro-retinitis may develop. Hysterical symptoms occasionally occur in girls. Epilepsy is not uncommon, and in fits developing in the adult the possibility of lead-poisoning should always be considered. An acute delirium may occur with hallucinations. The patients may have trance-like attacks, which follow or alternate with convulsions. A few cases of lead encephalopathy finally drift into lunatic asylums. Tremor is one of the commonest manifestations of lead-poisoning.

(*f*) *Arterio-sclerosis.*—Lead-workers are notoriously subject to arterio-sclerosis with contracted kidneys and hypertrophy of the heart. The cases usually show distinct gouty deposits, particularly in the big-toe joint; but in this country acute gout in lead-workers is rare. According to Sir William Roberts, the lead favors the precipitation of the crystalline urates of the tissues. Ralfe has shown that lead diminishes the alkalinity of the blood, and so lessens the solubility of the uric acid.

Prognosis.—In the minor manifestations of lead-poisoning this is good. According to Gowers, the outlook is bad in the primary atrophic form of paralysis. Convulsions are, as a rule, serious, and the mental symptoms which succeed may be permanent. Occasionally the wrist-drop persists.

Treatment.—Prophylactic measures should be taken at all lead-works, but unless employés are careful poisoning is apt to occur even under the most favorable conditions. Cleanliness of the hands and of the finger-nails, frequent bathing, and the use of respirators when necessary, should be insisted upon. When the lead is in the system, the iodide of potassium should be given in from five- to ten-grain doses three times a day. For the colic, local applications and, if severe, morphia may be used. An occasional morning purge of sulphate of magnesia may be given. For the anæmia iron should be used. In the very acute cases it is well not

to give the iodide, as, according to some writers, the liberation of the lead which has been deposited in the tissues may increase the severity of the symptoms. For the local palsies massage and the constant current should be used.

IV. ARSENICAL POISONING.

Acute poisoning by arsenic is common, particularly by Paris green and such mixtures as "Rough on Rats," which are used to destroy vermin and insects. The chief symptoms are intense pain in the stomach, vomiting, and, later, colic, with diarrhœa and tenesmus; occasionally the symptoms are those of collapse. If recovery takes place, paralysis may follow. The treatment should be similar to that of other irritant poisons—rapid removal with the stomach pump, the promotion of vomiting, and the use of milk and eggs. If the poison has been taken in solution, dialyzed iron may be used in large doses of from six to eight drachms.

Chronic Arsenical Poisoning.—Arsenic is used extensively in the arts, particularly in the manufacture of colored papers, artificial flowers, and in many of the fabrics employed as clothing. The glazed green and red papers used in kindergartens also contain arsenic. It is present also in many wall-papers and carpets. Much attention has been paid to this question of late years, as instances of poisoning have been thought to depend upon wall-papers and other household fabrics. According to J. J. Putnam, the greatest danger is from the dust blown off by currents of air or detached by the brush. It is thought, too, that possibly some volatile compound of arsenic may be formed. Arsenic is eliminated in all the secretions, and has been found in the milk. J. J. Putnam, it should be remembered, has shown that it is not uncommon to find traces of arsenic in the urine of many persons in apparent health. The effects of moderate quantities of arsenic are not infrequently seen in medical practice. In chorea and in pernicious anæmia, steadily increasing doses are often given until the patient takes from fifteen to twenty drops of Fowler's solution three times a day. Flushing and hyperæmia of the skin, puffiness of the eyelids or above the eyebrows, nausea, vomiting, and diarrhœa are the most common symptoms. Redness and sometimes bleeding of the gums and salivation occur. In the protracted administration of arsenic patients may complain of numbness and tingling of the fingers. In the large number of patients to whom I have administered arsenic, often in doses which might be termed excessive, I have seen only one case in which numbness and tingling were marked. Pigmentation of the skin I have seen on several occasions.

In the slow poisoning by the absorption of arsenic in minute doses, as from wall-paper and fabrics, the symptoms are varied. J. J. Putnam groups them into the cases in which the symptoms mainly concern the general nutrition without signs of local irritation; those in which the symptoms

are due to irritation of the conjunctivæ, mouth, or pharynx; those with symptoms pointing to the digestive tract; cases with marked nervous phenomena; and those in which the nutrition of some special part of the body is involved. The most common symptoms are those of anæmia and debility, perhaps with slight irritation of the mucous membrane, and numbness and tingling. How far these symptoms are to be attributed to the small quantities of arsenic absorbed from wall-papers and fabrics is by some considered doubtful. That children and adults may take with impunity large doses for months without unpleasant effects, and the fact of the gradual establishment of a toleration which enables Styrian peasants to take as much as eight grains of arsenious acid in a day, speak strongly against it.

Arsenical paralysis has the same characteristics as lead-palsy, but the legs are more affected than the arms, particularly the extensors and peroneal group, so that the patient has the characteristic *steppage* gait of peripheral neuritis.

The electrical reaction in the muscles may be disturbed before any loss of power, and when the patient is asked to extend the wrist fully and to spread the fingers slight weakness may be detected early.

V. PTOMAINE POISONING.

In the bacterial decomposition of animal matters chemical compounds are formed, the putrefactive alkaloids, known as ptomaines and toxines, some of which are highly poisonous. They differ extraordinarily in their chemical characters and physiological effects. Some only are poisonous, and these Brieger has designated as toxines. The specific action of the micro-organisms in disease is now attributed in large part to the formation of these bodies, and the whole question of immunity and protection is now being worked out in this direction, a special stimulus having been given of late in the discovery by Hankin of the so-called defensive alkaloids (see under pneumonia).

Our interest here is in the effects of these poisons when taken with foods.*

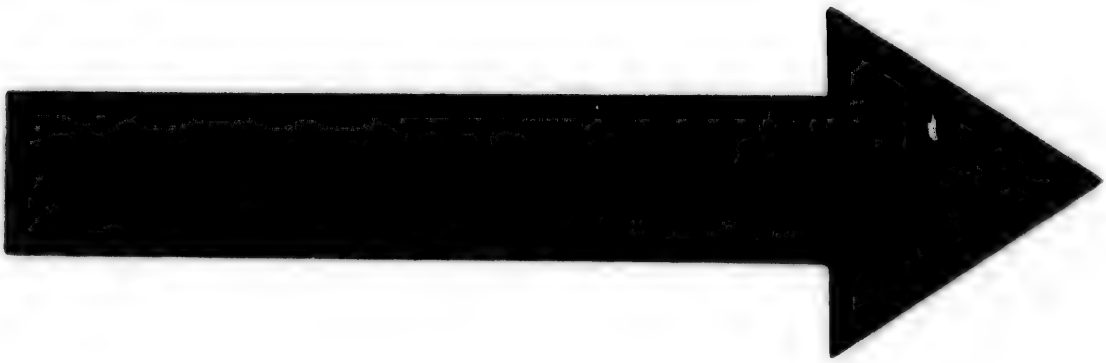
It is quite possible that the leucomaines, the basic substances formed in the living body, may under certain circumstances be capable of causing disease. Products also of the bacterial decomposition in the intestines may be absorbed and act as poisons. Our knowledge on these points is as yet scanty and uncertain. A suggestive chapter (XIII) upon the subject is to be found in the work of Vaughan and Novy.

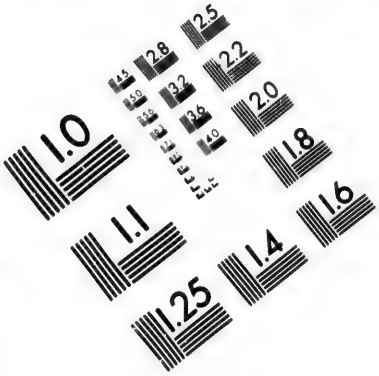
* For a full discussion of the whole subject the student is referred to the Manual upon Ptomaines and Leucomaines, by Vaughan and Novy, second edition, Philadelphia, 1891.

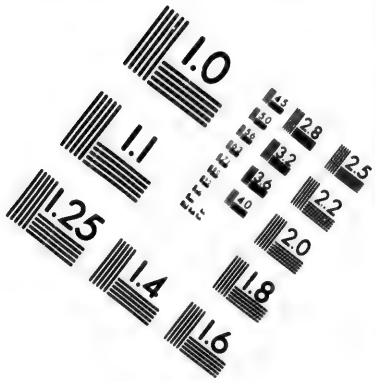
Among the more common forms are the following:

(1) **Meat Poisoning.**—Cases have usually followed the eating of sausages or pork-pie or head-cheese, and also occasionally beef, veal, and mutton. Sausage poisoning, which is known by the name of *botulism* or *allantiasis*, has long been recognized, and there have been numerous outbreaks, particularly in parts of Germany. Similar attacks have been produced by ham and by head-cheese. The precise nature of the poison in these cases has not yet been determined. Other outbreaks have followed the eating of beef and veal. In the majority of these cases the meat has undergone decomposition, though the change may not have been evident to the taste. The symptoms of meat poisoning are those of acute gastro-intestinal irritation. Ballard's description of the Wellbeck cases, quoted by Vaughan, holds good for a majority of them:

"A period of incubation preceded the illness. In 51 cases where this could be accurately determined, it was twelve hours or less in 5 cases; between twelve and thirty-six hours in 34 cases; between thirty-six and forty-eight hours in 8 cases; and later than this in only 4 cases. In many cases the first definite symptoms occurred suddenly, and evidently unexpectedly, but in some cases there were observed during the incubation more or less feeling of languor and ill-health, loss of appetite, nausea, or fugitive, griping pains in the belly. In about a third of the cases the first definite symptom was a sense of chilliness, usually with rigors, or trembling, in one case accompanied by dyspnœa; in a few cases it was giddiness with faintness, sometimes accompanied by a cold sweat and tottering; in others the first symptom was headache or pain somewhere in the trunk of the body—e. g., in the chest, back, between the shoulders, or in the abdomen, to which part the pain, wherever it might have commenced, subsequently extended. In one case the first symptom noticed was a difficulty in swallowing. In two cases it was intense thirst. But however the attack may have commenced, it was usually not long before pain in the abdomen, diarrhœa, and vomiting came on, diarrhœa being of more certain occurrence than vomiting. The pain in several cases commenced in the chest or between the shoulders, and extended first to the upper and then to the lower part of the abdomen. It was usually very severe indeed, quickly producing prostration or faintness, with cold sweats. It was variously described as crampy, burning, tearing, etc. The diarrhœal discharges were in some cases quite unrestrainable, and (where a description of them could be obtained) were said to have been exceedingly offensive and usually of a dark color. Muscular weakness was an early and very remarkable symptom in nearly all the cases, and in many it was so great that the patient could only stand by holding on to something. Headache, sometimes severe, was a common and early symptom; and in most cases there was thirst, often intense and most distressing. The tongue, when observed, was described usually as thickly coated with a brown, velvety fur, but red at the tip and edges. In the early stage the skin was often cold to the







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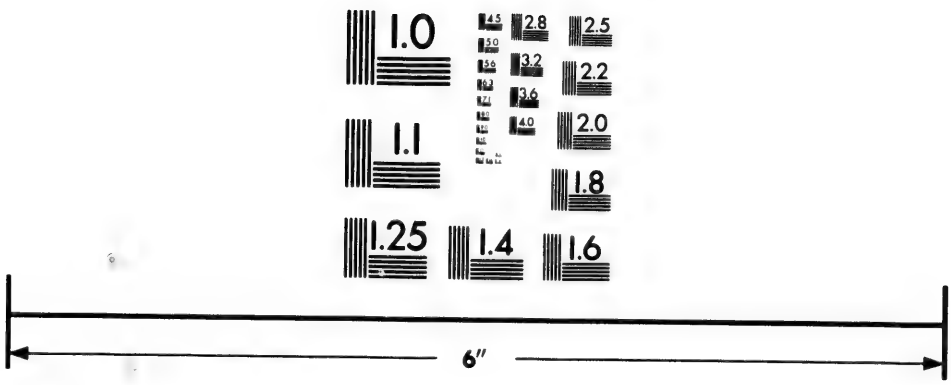

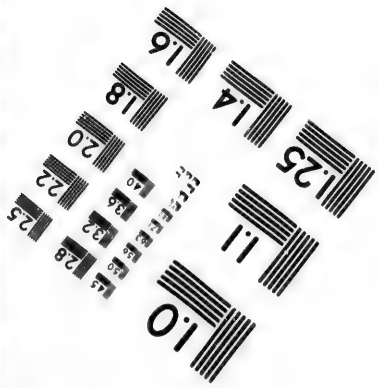

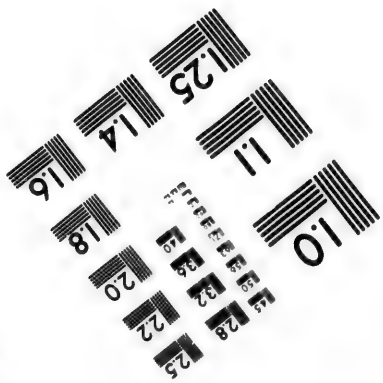

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touch, but afterward fever set in, the temperature rising in some cases to 101°, 103°, and 104° F. In a few severe cases, where the skin was actually cold, the patient complained of heat, insisted on throwing off the bed-clothes, and was very restless. The pulse in the height of the illness became quick, counting in some cases 100 to 128. The above were the symptoms most frequently noted. Other symptoms occurred, however, some in a few cases, and some only in solitary cases. These I now proceed to enumerate. Excessive sweating, cramps in the legs, or in both legs and arms, convulsive flexion of the hands or fingers, muscular twitchings of the face, shoulders, or hands, aching pain in the shoulders, joints, or extremities, a sense of stiffness of the joints, prickling or tingling or numbness of the hands lasting far into convalescence in some cases, a sense of general compression of the skin, drowsiness, hallucinations, imperfection of vision, and intolerance of light. In three cases (one that of a medical man) there was observed yellowness of the skin, either general or confined to the face and eyes. In one case, at a late stage of the illness, there was some pulmonary congestion and an attack of what was regarded as gout. In the fatal cases death was preceded by collapse like that of cholera, coldness of the surface, pinched features, and blueness of the fingers and toes and around the sunken eyes. The debility of convalescence was in nearly all cases protracted to several weeks.

"The mildest cases were characterized usually by little remarkable beyond the following symptoms, viz., abdominal pains, vomiting, diarrhœa, thirst, headache, and muscular weakness, any one or two of which might be absent."

Many instances are on record of poisoning by canned goods, particularly meat. Some of these, according to John G. Johnson, have been cases of corrosive poisoning from muriate of zinc and muriate of tin used as an amalgum, but poisonous effects identical with those just described have followed the use of canned meats.

Certain game birds, particularly the grouse, are stated to be poisonous, in special districts and at certain seasons of the year.

(2) **Poisoning by Milk Products.**—Poisoning by cheese has long been known. In Michigan, in 1883 and 1884, there were nearly 300 cases of cheese poisoning, and from pieces of the cheese Vaughan separated a substance which he called tyrotoxicon. Since that date other outbreaks have been reported. Apparently to this poison also are due the outbreaks following the use of milk, several of which are reported in the manual by Vaughan and Novy. Still more numerous of late years have been the cases due to poisonous ice-cream, in which also the tyrotoxicon has been found.

The symptoms are those of acute gastro-intestinal irritation, and are similar to those already detailed by Ballard.

(3) **Poisoning by Shell-fish and Fish.**—Perhaps the most serious form of *ichthysmus*, as the disease is called, is that produced by the mussel,

many epidemics of which have been studied of late, more particularly an outbreak at Wilhelmshaven. Brieger has separated a poison which he has called *mytilotoxin*. It has been shown that this exists chiefly in the liver of the mussel. It does not yet appear to be settled whether there is a special poisonous variety or whether the mussel only becomes toxic under certain conditions. The latter seems to be the most probable view, as Schmidtmann found that the non-poisonous mussels soon became toxic when placed in the Wilhelmshaven bay, while those from the bay soon lost their toxic properties when placed in the open sea.

The symptoms of mussel poisoning follow the eating of either raw or cooked mussels. The symptoms are those of an acute poisoning with profound action on the nervous system, and without gastro-intestinal symptoms. There are numbness and coldness, no fever, dilated pupils, rapid pulse, and death occurs sometimes within two hours with collapse symptoms.

Poisoning occasionally follows the eating of oysters which are stale or decomposed. The symptoms are usually gastro-intestinal. Certain fish also cause poisoning, more particularly the salted sturgeon used in parts of Russia, which has sometimes proved fatal to large numbers of persons. In the middle parts of Europe the barb is stated to be sometimes poisonous, producing the so-called "*barben cholera*." In China and Japan various species of the *tetrodon* are also toxic, sometimes proving fatal within an hour, with symptoms of intense disturbance of the nervous system. Several other poisonous forms are known, which produce symptoms described as *ichthysmus paralyticus*.

VI. GRAIN POISONING.

(1) Ergotism.—The prolonged use of meal made from grains contaminated with the ergot fungus (*claviceps purpurea*) causes a series of symptoms known as ergotism, epidemics of which have prevailed in different parts of Europe. Two forms of this chronic ergotism are described—the gangrenous and the convulsive or spasmodic. In the former, mortification affects the extremities—usually the toes and fingers, less commonly the ears and nose. Preceding the onset of the gangrene there are usually anæsthesia, tingling, pains, spasmodic movements of the muscles, and gradual blood stasis in certain vascular territories.

The nervous manifestations are very remarkable. After a prodromal stage of ten to fourteen days, in which the patient complains of weakness, headache, and tingling sensations in different parts of the body, perhaps accompanied with slight fever, spasmodic symptoms develop, producing cramps in the muscles and contractures. The arms are flexed and the legs and toes extended. These spasms may last from a few hours to many days and relapses are frequent. In severer cases epilepsy develops and the

patient may die in convulsions. Mental symptoms are common, manifested sometimes in a preliminary delirium, but more commonly, in the chronic poisoning, as melancholia or dementia. Posterior spinal sclerosis occurs in chronic ergotism. In the interesting group of 29 cases studied by Tuczek and Siemens, nine died at various periods after the infection, and four post-mortems showed degeneration of the posterior columns. A condition similar to tabes dorsalis is gradually produced by this slow degeneration in the spinal cord.

(2) **Lathyrism** (*Lupinosis*).—An affection produced by the use of meal from varieties of vetches, chiefly the *Lathyrus sativus* and *L. cicera.* The grain is popularly known as the chick-pea. The grains are usually powdered and mixed with the meal from other cereals in the preparation of bread. As early as the seventeenth century it was noticed that the use of flour with which the seeds of the *Lathyrus* were mixed caused stiffness of the legs. The subject did not, however, attract much attention until the studies of James Irving, in India, who between 1859 and 1868 published several important communications, describing a form of spastic paraplegia affecting large numbers of the inhabitants in certain regions of India and due to the use of meal made from the *Lathyrus* seeds. It also produces a spastic paraplegia in animals. The Italian observers describe a similar form of paraplegia, and it has been observed in Algiers by the French physicians. The condition is that of a spastic paralysis, involving chiefly the legs, which may proceed to complete paraplegia. The arms are rarely, if ever, affected. It is evidently a slow sclerosis induced under the influence of this toxic agent. The precise anatomical condition, so far as I can ascertain, has not yet been determined.

(3) **Pellagra.**—This is a nutritional disturbance due to the use of altered maize. The disease occurs extensively in parts of Italy, in the south of France, and in Spain. It has not been observed in this country. It prevails extensively among the poorer classes, particularly in the country districts, and appears to be associated in some way with the use of maize which (according to most authorities) is fermented or diseased. In the early stage the symptoms are indefinite, characterized by debility, pains in the spine, insomnia, digestive disturbances, more rarely diarrhœa. The first clear manifestation of the disease is the pellagral erythema, which almost invariably appears in the spring. This is followed by desiccation and exfoliation of the epidermis, which becomes very rough and dry, and occasionally crusts form, beneath which there is suppuration. With these cutaneous manifestations there are digestive troubles—salivation, dyspepsia, and diarrhœa—which may be of a dysenteric nature. After lasting for a few months improvement occurs in the milder cases and convalescence is gradually established. In the more severe and chronic forms there are pronounced nervous symptoms—headache, backache, spasms, and finally paralysis and mental disturbance. The paralytic condition affects the legs and leads gradually to paraplegia. The mental manifestations, which

are rarely met with until the third or fourth attack, are melancholia or suicidal mania. Finally, there may be a condition of the most pronounced cachexia.

The anatomical changes are indefinite. Chronic degenerative changes have been found, particularly fatty degeneration and a peculiar pigmentation in the viscera. The measures to be employed are change in diet, removal from the infected district, and, as a prophylaxis, proper preservation of the maize.*

VII. SUNSTROKE

(*Heat Exhaustion; Insolation; Thermic Fever; Heat-stroke; Coup de Soleil*).

Definition.—A condition produced by exposure to excessive heat.

It is one of the oldest of recognized diseases; two instances are mentioned in the Bible. It was long confounded with apoplexy. The Anglo-Indian surgeons gave admirable descriptions of it. In this country the most important contributions have come from the New York Hospital and the Pennsylvania Hospital; from the former, the studies of Swift and Darrach, from the latter, the papers of Gerhard, George B. Wood, the elder Pepper, and Levick. In New Orleans, Bennett Dowler studied the disease and recognized the difference between heat exhaustion and sunstroke. Very little has been added to our knowledge of the disease since the publication of a monograph by H. C. Wood. Two forms are recognized, heat exhaustion and heat-stroke.

Heat Exhaustion.—Prolonged exposure to high temperatures, particularly when combined with physical exertion, is liable to be followed by extreme prostration, collapse, restlessness, and in severe cases by delirium. The surface is usually cool, the pulse small and rapid, and the temperature may be subnormal—as low as 95° or 96°. The individual need not necessarily be exposed to the direct rays of the sun, but the condition may come on when working in close, confined rooms during midsummer. It may also follow exposure to great artificial heat; thus the stokers in the Atlantic steamships sometimes succumb to the effect of the great heat in the engine rooms.

Sunstroke or Thermic Fever.—The cases are chiefly found in persons who, while working very hard, are exposed to the sun. Soldiers on the march with their heavy accoutrements are particularly liable to attack. In the larger cities of this country the cases are almost exclusively confined to workmen who are much exposed and, at the same time, have been drinking beer and whisky.

Morbid Anatomy and Pathology.—*Rigor mortis* occurs early. Putrefactive changes develop with great rapidity. The venous engorge-

* The most elaborate discussion of the subject is by Jules Arnould in the Dictionnaire Encyclopédique des Sciences Médicales, tome xxii, 1886.

ment is extreme, particularly in the cerebrum. The left ventricle is contracted (Wood), and the right chamber dilated. The blood is usually fluid; the lungs are intensely congested. Parenchymatous changes occur in the liver and kidneys.

According to Wood, "heat exhaustion with lowered temperature represents a sudden vaso-motor palsy, i. e., a condition in which the existing effect of the heat paralyzes the centre in the medulla." On the other hand, thermic fever is held to be due to paralysis under the influence of the extreme external heat of the centre in the medulla which regulates the disposition of the bodily heat. Owing to this disturbance, more heat is produced and less given off than normally.

Symptoms.—The patient may be struck down and die within an hour with symptoms of heart failure, dyspnœa, and coma. This form, sometimes known as the asphyxial, occurs chiefly in soldiers and is graphically described by Parkes. Death indeed may be almost instantaneous, the victims falling as if struck upon the head. The usual form in this latitude comes on during exposure, with pain in the head, dizziness, a feeling of oppression, and sometimes nausea and vomiting. Visual disturbances are common, and a patient may have colored vision. Diarrhœa or frequent micturition may supervene. Insensibility follows, which may be transient or which deepens into a profound coma. The patients are usually admitted to hospital in an unconscious state, with the face flushed, the skin pungent, the pulse rapid and full, and the temperature ranging from 107° to 110°, or even higher. F. A. Packard states that of the 31 cases admitted to the Pennsylvania Hospital in the summer of 1887, in a majority of them the temperature was between 110° and 111°. In one case the temperature was 112°. The breathing is labored and deep, sometimes stertorous. Usually there is complete relaxation of the muscles, but twitchings, jactitation, or very rarely convulsions may occur. The pupils may at first be dilated, but by the time the cases are admitted to hospital they are (in a majority) extremely contracted. Petechiæ may be present upon the skin. In the fatal cases the coma deepens, the cardiac pulsations become more rapid and feeble, the breathing becomes hurried and shallow and of the Cheyne-Stokes type. The fatal termination may occur within twenty-four or thirty-six hours. Favorable indications are the recovery of consciousness and a fall in the fever. The recovery in these cases may be complete. In other instances there are remarkable after-effects, the most constant of which is a permanent inability to bear high temperatures. Such patients become very uneasy when the thermometer reaches 80° F. in the shade. An extraordinary instance came under my notice in which the patient was subsequently so sensitive to temperatures in the neighborhood of 75° F. that at such times he lived comfortably only in the cellar, and finally sought refuge in Alaska. Loss of the power of mental concentration and failure of memory are more constant and very troublesome sequelæ. Such patients are always worse

in the hot weather. Occasionally convulsions and marked mental disturbance may develop. H. C. Wood states that in a case of this kind chronic meningitis was found.

Guitéras has called attention to a form of fever occurring in the South, known in Florida as "Florida fever," in the Carolinas as "country fever," and in tropical countries as *fièvre inflammatoire* The cases last for a variable time, and are mistaken for malaria or typhoid; but he believes them to be entirely distinct and due to a prolonged action of the high temperatures. He has called the condition a "continued thermic fever."

The diagnosis of heat exhaustion from thermic fever is readily made, as the difference between the two conditions is striking. "In solar exhaustion the skin is moist, pale, and cool; the breathing is easy though hurried; the pulse is small and soft; the vital forces fall into a temporary collapse; the senses remain entire" (Dowler); whereas in sunstroke or heat apoplexy there is usually unconsciousness and pyrexia.

The mode of onset, together with the circumstances under which it occurs and the high temperature, permits thermic fever to be readily differentiated from apoplexy, and coma from other conditions.

Treatment.—In heat exhaustion stimulants should be given freely, and if the temperature is below normal the hot bath should be used Ammonia may be given if necessary. In thermic fever the indications are to reduce the temperature as rapidly as possible. This may be done by placing the patient in a bath at 70°. Rubbing the body with ice was practised at the New York Hospital by Darrach in 1857, and is an excellent procedure to lower the temperature rapidly. Ice-water enemata may also be employed. At the Pennsylvania Hospital in the summer of 1887 the ice-pack was used with great advantage. Of 31 cases only 12 died, a result probably as satisfactory as can be obtained, considering that many of the patients are almost moribund when brought to hospital. It should be compared with Swift's statistics, in which of 150 cases 78 died. In the cases in which the symptoms are those of intense asphyxia, and in which death may take place in a few minutes, free bleeding should be practised, a procedure which saved Weir Mitchell when a young man. Of other remedies, the antipyretics have been employed, and may be given when there is any special objection to hydrotherapy, for which, however, they cannot be substituted.

VIII. OBESITY.

Corpulence, an excessive development of the bodily fat, is a condition for which the physician is frequently consulted, and for which much may be done by a judicious arrangement of the diet. The tendency to polysarcia or obesity is often hereditary, and is particularly apt to be manifest after the middle period of life. It may, however, be seen early, and in this country it is not very uncommon in young girls and young boys.

A very important factor is overeating, a vice which is more prevalent and only a little behind overdrinking in its disastrous effects. A majority of persons over forty years of age habitually eat too much. In some of the most aggravated cases of obesity, however, this plays no part, and the unfortunate victim may be a notoriously small eater. A second element is lack of proper exercise; a third less important factor is the taking largely of alcoholic beverages, particularly beer.

In obesity it is now generally conceded that the carbohydrates, which were so long blamed, are not at fault, since they are themselves converted into water and carbon dioxide. On account, however, of the facility with which they are utilized for the purposes of oxidation the albuminous elements of the food are less readily oxidized, not so fully decomposed, and the fat is in reality separated from them. So, too, the fats themselves are not so prone to cause obesity as the carbohydrates, being less readily oxidized and interfering less with the complete metabolism of the albuminous elements.

Many plans are now advised for the reduction of fat, the most important of which are those of Banting, Ebstein, and Oertel. In the Banting method the amount of food is reduced, the liquids are restricted, and the fats and carbohydrates excluded.

Ebstein recommends the use of fat and the rapid exclusion of the carbohydrates. The following is an example of his dietary:

Breakfast (6 A. M. in summer, 7.30 A. M. in winter).—White bread, well toasted (rather less than two ounces) and well covered with butter. Tea, without milk or sugar, eight or nine ounces.

Dinner, 2 P. M.—Soup made with beef-marrow. Fat meat, with fat sauce, four to five ounces. A moderate quantity of asparagus, spinach, cabbage, peas, and beans. Two or three glasses of light white wine. After the meal, a large cup of tea without milk or sugar.

Supper, at 7.30 P. M.—An egg, a little roast meat, with fat. About an ounce of bread, well covered with butter. A large cup of tea, without milk or sugar.

Oertel's method has already been considered in connection with the treatment of fatty heart, and is combined with systematic bodily exercise. It is particularly adapted for stout persons with weak heart.

The so-called Schweninger cure is in reality Oertel's, with the sole modification of the forbidding of any fluid at meals. Liquids must be taken more than two hours after the food.

Yeo, after a full consideration of the various methods, gives the following useful summary:

"The albuminates in the form of animal food should be strictly limited. Farinaceous and all starchy foods should be reduced to a minimum. Sugar should be entirely prohibited. A moderate amount of fats, for the reasons given by Ebstein, should be allowed.

"Only a small quantity of fluid should be permitted at meals, but

enough should be allowed to aid in the solution and digestion of the food. Hot water or warm aromatic beverages may be taken freely between meals or at the end of the digestive process, especially in gouty cases, on account of their eliminative action.

"No beer, porter, or sweet wines of any kind to be taken; no spirit, except in very small quantity. It should be generally recognized that the use of alcohol is one of the most common provocatives of obesity. A little Hock, still Moselle, or light claret, with some alkaline table water is all that should be allowed. The beneficial effects of such diet will be aided by abundant exercise on foot and by the free use of saline purgatives, so that we may insure a complete daily unloading of the intestinal canal.

"It is only necessary to mention a few other details. Of animal foods, all kinds of lean meat may be taken, poultry, game, fish (eels, salmon, and mackerel are best avoided), eggs.

"Meat should not be taken more than once a day, and not more than six ounces of cooked meat at a time. Two lightly boiled or poached eggs may be taken at one other meal, or a little grilled fish.

"Bread should be toasted in thin slices and completely, not browned on the surface merely.

"Hard captain's biscuits may also be taken.

"Soups should be avoided, except a few tablespoonfuls of clear soup.

"Milk should be avoided, unless skimmed and taken as the chief article of diet. All milk and farinaceous puddings and pastry of all kinds are forbidden. Fresh vegetables and fruit are permitted.

"It is important to bear in mind that the actual quantity of food permitted must have a due relation to the physical development of the individual, and that what would be adequate in one case might be altogether inadequate in the case of another person of larger physique." *

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* A System of Therapeutics, vol. i, edited by H. A. Hare, Philadelphia, 1891.

SECTION XI.

DISEASES DUE TO ANIMAL PARASITES.

I. PSOROSPERMIASIS.

UNDER this term are embraced several affections produced by the sporozoa. These parasites belonging to the lowest division of the protozoa, are also known as psorosperms and gregarinidæ. They are extraordinarily abundant in the invertebrates, and are not uncommon in the higher mammals. The entire group of blood parasites, hæmatozoa, which live within the corpuscles, are closely related to them. Psorosperms are, as a rule, parasites of the cells—*cytozoa*. The commonest and most suitable variety for study is the *coccidium oviforme* of the rabbit, which produces a disease of the liver in which the organ is studded throughout with whitish nodules, ranging in size from a pin's head to a split pea. On section each nodule is seen to be a dilated portion of a bile duct; the walls are lined with epithelium in the interior of which are multitudes of ovoid bodies—the coccidia. Another very common form occurs in the muscles of the pig, the so-called Rainey's tube, which is an ovoid body within the sarcolemma containing a number of small, sickle-shaped, unicellular organisms.

These bodies probably play a more important *rôle* in human pathology than has hitherto been thought. The cases reported may be grouped under the following divisions: internal and external.

(1) **Internal Psorospermiasis.**—In a majority of the cases of this group the psorosperms have been found in the liver, producing a disease similar to that which occurs in rabbits. In Guebler's case there were tumors which could be felt in the liver during life, and they were determined by Leuckart to be due to coccidia. In W. B. Haddon's case the patient was admitted to St. Thomas's Hospital with slight fever, drowsiness, and gradual unconsciousness; death occurred on the fourteenth day of observation. Whitish neoplasms were found upon the peritonæum, omentum, and on the layers of the pericardium; and a few were found in the liver, spleen, and kidneys. A somewhat similar case, though more remarkable, as it ran a very acute course, is reported by Silcott. A woman, aged fifty-three, admitted to St. Mary's Hospital, was thought to be suffering from typhoid

fever. She had had a chill six weeks before admission. There was fever of an intermittent type, slight diarrhœa, nausea, tenderness over the liver and spleen, and a dry tongue; death occurred from heart-failure. The liver was enlarged, weighed eighty-three ounces, and in its substance there were caseous foci, around each of which was a ring of congestion. The spleen weighed sixteen ounces and contained similar bodies. The ileum presented six papule-like elevations. The masses resembled tubercles, but on examination coccidia were found.

The parasites are also found in the kidneys and ureters. Cases of this kind have been recorded by Bland Sutton and Paul Eve. In the case reported by Eve the symptoms were hæmaturia and frequent micturition, and death took place on the seventeenth day. The nodules throughout the pelvis and ureters have been regarded as mucous cysts. In a case reported by Joseph Griffiths the tumors in the ureter caused hydronephrosis.

(2) **Cutaneous Psorospermiasis.**—(*a*) *Follicular.*—This remarkable skin-disease was originally described by J. C. White, under the name of *keratosis follicularis.* Darier, of Paris, has shown that this is really a parasitic affection. The lesions are chiefly on the face, the flanks, and the inguinal regions. It is at first papular, surmounted by a grayish crust, dry and hard. The lesions finally become confluent, and form a series of irregular elevations giving a rasp-like feeling to the touch.

Microscopical examination shows that in these papillomatous growths there are numerous organisms corresponding to psorosperms. At the St. Louis Hospital, in Paris, Darier was kind enough to show me the cases and the specimens from them. No one accustomed to the appearance of psorosperms as seen in the lower animals could question the truly parasitic nature of these bodies. A case of the disease has been reported in this country by A. R. Robinson.

(*b*) *Paget's Disease of the Nipple.*—In this affection, formerly regarded as an eczema, psorosperms are constantly present, as shown by Darier, A. B. Macallum, and others. They are readily demonstrated, without any special preparation, and here, too, of the nature of the bodies there can be no question.

In molluscum contagiosum and in epithelioma many observers have noted the presence of bodies which lie in and between the epithelial cells and have some resemblance to psorosperms. The bodies are readily seen in sections of epithelioma, but they lack the sharply defined characters of the coccidia which are present in Paget's disease and in White's keratosis.

II. DISTOMIASIS.

Several forms of trematodes or flukes are parasitic in man, and when in numbers may cause serious disease.

(1) *Liver Flukes.*—The following varieties of flukes have been found: The *distoma hepaticum*, a very common parasite in ruminants, which has a length of from twenty-eight to thirty-two millimetres. The *distoma lanceolatum*, a much smaller form, from eight to ten millimetres in length, which is also very common in sheep and cattle. The *distoma crassum*, the largest form, measuring from four to eight centimetres in length. One or two other less important forms have occasionally been met with. The studies of the Japanese physicians have brought to light the interesting fact that there is a distoma widely endemic in certain provinces in that country. Two forms have been described, the *distoma endemicum* and the *distoma perniciosum*, about which there is still a doubt whether they are different species or not. The studies of Ijima indicate that they are probably the same. According to Baelz, fully twenty per cent of the inhabitants of certain provinces are affected.

The flukes occupy the bile-passages and the upper portion of the small intestine. When in large numbers they may cause serious and fatal disease of the liver, usually with ascites and jaundice. The liver may be enormously enlarged; in Kichner's case it weighed eleven pounds. The flukes may cause a chronic cholangitis, leading to great thickening or even calcification of the walls of the bile-duct.

The endemic fluke disease of Japan is characterized by enlargement of the liver, emaciation, diarrhœa, and frequently ascites.

(2) *The Blood Fluke; Bilharzia Hæmatobia.*—This trematode is found in Egypt, southern Africa, and Arabia, and is the cause in these countries of the endemic hæmaturia. The female is about two centimetres in length, cylindrical, filiform, and about ·07 millimetre in diameter. The parasite lives in the venous system, particularly in the portal vein, and in the veins of the spleen, bladder, kidneys, and mesentery. According to Bilharz, at least fifty per cent of the lower classes in Egypt are infected with it. It is not yet known how the parasite gains entrance to the body. In all probability it is by drinking impure water containing the embryos.

The symptoms are due to changes in the mucous membrane of the urinary organs caused by the presence of the parasites in the blood-vessels of these parts. Hæmaturia is the first and most constant symptom, leading gradually to anæmia. There is generally pain during micturition. The blood is not constant in the urine. The ova of the Bilharzia are readily seen under a microscope with a low power. They are ovoid in shape, translucent, with a small spike at one end. The embryo can be readily seen.

The disease is rarely fatal; a great majority of the cases recover. Chil-

dren are more commonly attacked than grown persons, and the disease often disappears by the time of puberty.

(3) *Bronchial Fluke; Distoma Ringeri; Parasitic Hæmoptysis.*—In parts of China, Japan, and Formosa there is an epidemic disease, described by Ringer and Manson, characterized by attacks of cough and hæmoptysis associated with the presence of a small fluke in the bronchial tubes.

III. DISEASES CAUSED BY NEMATODES.

I. Ascariasis.

(*a*) *Ascaris lumbricoides*, the most common human parasite, is found chiefly in children. The female is from seven to twelve inches in length, the male from four to eight inches. The worm is cylindrical, pointed at both ends, and has a yellowish-brown, sometimes a slightly reddish color. Four longitudinal bands can be seen, and it is striated transversely. The ova, which are sometimes found in large numbers in the fæces, are small, brownish-red in color, elliptical, and have a very thick covering. They measure ·075 millimetre in length and ·058 millimetre in width. They develop outside the body, but the life history is not known. The parasite occupies the upper portion of the small intestine. Usually not more than one or two are present, but occasionally they occur in enormous numbers. The migrations are peculiar. They may pass into the stomach, from which they may be ejected by vomiting, or they may crawl up the œsophagus and enter the pharynx, from which they may be withdrawn. A child, under my care in the small-pox department of the General Hospital, during convalescence, withdrew in this way more than thirty round worms within a few weeks. In other instances the worm passes into the larynx, and has been known to cause fatal asphyxia, or passing into the trachea, to cause gangrene of the lung. They may pass into the Eustachian tube and appear at the external meatus. The most serious migration is into the bile-duct. There is a specimen in the Wistar-Horner Museum of the University of Pennsylvania in which not only the common duct, but also the main branches throughout the liver are enormously distended and packed with numerous round worms. The bowel may be perforated by them and peritonitis result.

The symptoms are not definite. When a few are present they may be passed without causing disturbance. In children there are irritative symptoms usually attributed to worms, such as restlessness, irritability, picking at the nose, grinding of the teeth, twitchings, or convulsions. These symptoms may be marked in very nervous children.

Treatment.—Santonin can be given, mixed with sugar, in doses of from one to three grains for a child and three to five grains for an adult, followed by a calomel or a saline purge. The dose may be given for

three or four days. An unpleasant consequence which sometimes follows the administration of this drug is xanthopsia or yellow vision.

(*b*) *Oxyuris Vermicularis* (*Thread-worm; Pin-worm*).—This common parasite occupies the rectum and colon. The male measures about four millimetres in length, the female about ten millimetres. They produce great irritation and itching, particularly at night, symptoms which become intensely aggravated by the nocturnal migration of the parasites.

The patients become extremely restless and irritable, the sleep is often disturbed, and there may be loss of appetite and anæmia. Though most common in children the parasite occurs at all ages.

The worm is readily detected in the fæces. Infection probably takes place through the water or possibly through salads, such as lettuces and cresses. A person the subject of the worms passes ova in large numbers in the fæces, and the possibility of reinfection must be scrupulously guarded against.

The treatment is simple, though occasionally there are instances in which all forms of medication are resisted. A case is mentioned of a gentleman, aged forty, who had suffered from childhood and had failed to obtain any benefit from prolonged treatment by many helminthologists. Santonin may be used in small doses, and mild purgatives, particularly rhubarb. Large injections containing carbolic acid, vinegar, quassia, aloes, or turpentine may be employed. In children the use of cold injections of strong salt and water is usually efficacious. They should be repeated for at least ten days. In giving the injection care should be taken to have the hips well elevated so that the fluid can be retained as long as possible. For the intense itching and irritation at night vaseline may be freely used or belladonna ointment.

II. Trichiniasis.

The trichina spiralis in its adult condition lives in the small intestine. The disease is produced by the embryos, which pass from the intestines and reach the voluntary muscles, where they finally become encapsulated —muscle trichinæ. It is in the migration of the embryos that the group of symptoms known as trichiniasis is produced.

Description of the Parasites.—(*a*) Adult or intestinal form. The female measures from three to four millimetres; the male, 1·5 millimetre, and has two little projections from the hinder end.

(*b*) The embryo or muscle trichina is from 0·6 to one millimetre in length and lies coiled in an ovoid capsule, which is at first translucent, but subsequently opaque and infiltrated with lime salts. The worm presents a pointed head and a somewhat rounded tail.

When flesh containing the trichinæ is eaten by man or by any animal in which the development can take place, the capsules are digested and the trichinæ set free. They pass into the small intestine, and about

the third day attain their full growth and become sexually mature. Virchow's experiments have shown that on the sixth or seventh day the embryos are fully developed. The young produced by each female trichina have been estimated at several hundred. Leukart thinks that various broods are developed in succession, and that as many as a thousand embryos may be produced by a single worm. The time from the ingestion of the flesh containing the muscle trichinæ to the development of the brood of embryos in the intestines is from seven to nine days. As soon as born the embryo trichinæ leave the intestines; wandering through the peritonæum and the connective tissues, probably through the mesentery and retroperitoneal tissues—some hold by means of the blood current—they finally reach the muscles, which constitute "the seat of election." After a preliminary migration in the intermuscular connective tissue they penetrate the primitive muscle fibres, and in about two weeks develop into the full-grown muscle form. In this process an interstitial myositis is excited and gradually an ovoid capsule develops about the parasite. Two, occasionally three or four, worms may be seen within a single capsule. This process of encapsulation has been estimated to take about six weeks. Within the muscles the parasites do not undergo further development. Gradually the capsule becomes thicker, and ultimately lime salts are deposited within it. This change may take place in man within four or five months. In the hog it may be deferred for many years. The calcification renders the cyst visible, and since first seen by Tiedemann, in 1822, and Hilton, in 1832, these small, opaque, oat-shaped bodies have been familiar objects to demonstrators of normal and morbid anatomy. The trichinæ may live within the muscles for an indefinite period. They have been found alive and capable of developing as late as twenty or even twenty-five years after their entrance into the system. In many instances, however, the worms are completely calcified. The trichina occurs in swine, in the rat, occasionally in mice and cats; it has been found also in the fox and a few other animals. The parasite was first found in the hog by the late Joseph Leidy. Experimentally, guinea-pigs and rabbits are readily infected by feeding them with muscle containing the larval form. Dogs are infected with difficulty; cats more readily. Experimentally, animals sometimes die of the disease if large numbers of the parasites have been eaten. In the hog the trichinæ, like the cysticerci, cause few if any symptoms. An animal the muscles of which are swarming with living trichinæ may be well nourished and healthy-looking. An important point also is the fact that in the hog the capsule does not readily become calcified, so that the parasites are not visible as in the human muscles. For a long time the trichina was looked upon as a pathological curiosity, but in 1860 Zenker discovered in a girl in the Dresden Hospital who had symptoms of typhoid fever both the intestinal and the muscle forms of the trichinæ, since which time the disease has been thoroughly studied.

Man is infected by eating the flesh of trichinous hogs. The incidence of the disease in swine varies much in different countries. In Germany, where a thorough and systematic microscopic examination of all swine flesh is made, the proportion of trichinous hogs is about 1 in 1,852. At the Berlin abattoir, where the microscopic examination is conducted by a staff of over eighty men and women, two portions are taken from the abdominal muscles, from the diaphragm, and from the intercostal muscles, and one piece from the muscles of the larynx and tongue. A special compressor is used to flatten the fragments of the muscle, and the examination is made with a magnifying power of from seventy to one hundred diameters. During the three years ending in 1885 there were 603 trichinous hogs detected, a ratio of 1 to 1,292. Statistics are not available in England. In the United States systematic inspection is unknown, and the statistics are by no means extensive enough. "Taking all the examinations of American pork thus far made, both at home and abroad, and we have a total of 298,782, in which trichinæ were found 6,280 times, being 2·1 per cent, or 1 to 48" (Salmon, 1884).

In 1883, in conjunction with A. W. Clement, I examined 1,000 hogs at the Montreal abattoir, and found only 4 infected. There is no reason to believe that the hog of this country is less liable to trichina than the German animal.

Modes of Infection.—The danger of infection depends entirely upon the mode of preparation of the flesh. Thorough cooking, so that all parts of the meat reach the boiling point, destroys the parasites; but in large joints the central portions are often not raised to this temperature. The frequency of the disease in different countries depends largely upon the habits of the people in the preparation of pork. In North Germany, where raw ham and *wurst* are freely eaten, the greatest number of cases have occurred. In South Germany, France, and England cases are rare. In this country the greatest number of persons attacked have been Germans. Salting and smoking the flesh are not always sufficient, and the Havre experiments showed that animals are readily infected when fed with portions of the pickled or the smoked meat as prepared in this country. Carl Fränkel, however, states that the experiments on this point have been negative, and that it is very doubtful if any cases of trichiniasis in Germany have been caused by American pork.

Frequency of Infection.—The dissecting-room and post-mortem statistics show that from one half to two per cent of all bodies contain trichinæ. Of 1,000 consecutive autopsies of which I have notes the trichinæ were present in 6 instances. I have, in addition, seen them in two dissecting-room cases and in two bodies at the Philadelphia Hospital.

The disease often occurs in epidemics, a large number of persons being infected from a single source. Among the best known of these outbreaks are the Hedersleben, in which there were 337 persons affected, and the Emersleben, in which there were 250 persons attacked. The extensive

outbreaks of this sort have been, with few exceptions, in North Germany. Alfred Mann, after a careful search, at my request, of the literature in the Surgeon-General's library, finds records of 456 cases in this country. The two largest groups of cases were at Astoria, Ore., reported by Kinney, 15 cases and one death; and at St. Paul, Minn., reported by Persons and Andrews, 15 cases and three deaths.

Symptoms.—The ingestion of trichinous flesh is not necessarily followed by the disease. When a limited number are eaten only a few embryos pass to the muscles and may cause no symptoms. Well-characterized cases present a gastro-intestinal period and a period of general infection.

In the course of a few days after eating the infected meat there are signs of gastro-intestinal disturbance—pain in the abdomen, loss of appetite, vomiting, and sometimes diarrhœa. The preliminary symptoms, however, are by no means constant, and in some of the large epidemics cases have been observed in which they have been absent. In other cases the gastro-intestinal features have been marked from the outset, and the attack has resembled cholera nostras. Pains in different parts of the body, general debility, and weakness have been noted in some of the epidemics.

The invasion symptoms develop between the seventh and the tenth day, sometimes not until the end of the second week. There is fever, except in very mild cases Chills are not common. The thermometer may register 102° or 104°, and the fever is usually remittent or intermittent. The migration of the parasites in the muscles excites a more or less intense myositis, which is characterized by pain on pressure and movement, and by swelling and tension of the muscles. The limbs are placed in the positions in which the muscles are in least tension. The involvement of the muscles of mastication and of the larynx may cause difficulty in chewing and swallowing. In severe cases the involvement of the diaphragm and intercostal muscles may lead to intense dyspnœa, which sometimes proves fatal. Œdema, a feature of great importance, may be early in the face. Later it develops in the extremities when the swelling and stiffness of the muscles are at their height. Profuse sweats, tingling and itching of the skin, and in some instances urticaria, have been described. The general nutrition is much disturbed and the patient becomes emaciated and often anæmic, particularly in the protracted cases. The patellar tendon reflex may be absent. The patients are usually conscious, except in cases of very intense infection, in which the delirium, dry tongue, and tremors give a picture similar to typhoid fever. In addition to the dyspnœa, present in the severer cases, there may be bronchitis, and in the fatal cases pneumonia or pleurisy. In some epidemics polyuria has been a common symptom. Albuminuria is frequent.

The intensity and duration of the symptoms depend entirely upon the grade of infection. In the mild cases recovery is complete in from ten to fourteen days. In the severe forms convalescence is not established for six or eight weeks, and it may be months before the patient recovers the

muscular strength. One case in the Hedersleben epidemic was weak eight years after the attack.

Of 72 fatal cases in the Hedersleben epidemic the greatest mortality occurred in the fourth and fifth and sixth weeks; namely, 52 cases. Two died in the second week with severe choleraic symptoms.

The mortality has ranged in different outbreaks from one or two per cent to thirty per cent. In the Hedersleben epidemic 101 persons died. Among the 456 cases reported in this country there were 122 deaths.

The *anatomical changes* are chiefly in the voluntary muscles. In the early stages they look normal, but in the fourth or fifth week grayish-white areas appear in which the muscle fibres are extensively degenerated and in the neighborhood of the trichinæ there is an acute interstitial myositis. Cohnheim has described a fatty degeneration of the liver and enlargement of the mesenteric glands. At the time of death in the fourth or fifth week or later the adult trichinæ are still found in the intestines.

The *prognosis* depends much upon the quantity of infected meat which has been eaten and the number of trichinæ which mature in the intestines. In children the outlook is more favorable. Early diarrhœa and moderately intense gastro-intestinal symptoms are, as a rule, more favorable than constipation.

Diagnosis.—This is perfectly clear when a large number of persons are infected at once and the parasites have been found in the ham or sausages. The worms may be discovered in the stools. The stools should be spread on a glass plate or black background and examined with a low-power lens, when the trichinæ are seen as small, glistening, silvery threads. In doubtful cases the diagnosis may be made by the removal of a small fragment of muscle. A special harpoon has been devised for this purpose by means of which a small portion of the biceps or of the pectoral muscle may be readily removed. Under cocaine anæsthesia an incision may be made and a small fragment removed. The disease may be mistaken for acute rheumatism, particularly as the pains are so severe on movement, but there is no special swelling of the joints. The tenderness is in the muscles both on pressure and on movement. The intensity of the gastro-intestinal symptoms in some cases has led to the diagnosis of cholera. Many of the former epidemics were doubtless described as typhoid fever, which the severer cases, owing to the prolonged fever, the sweats, the delirium, dry tongue, and gastro-intestinal symptoms, somewhat resemble. The pains in the muscles, swelling, œdema, and shortness of breath are the most important diagnostic points. Under acute myositis reference has already been made to the cases which closely resemble trichiniasis. The epidemic in 1879 on board the training ship Cornwall presented symptoms similar to those of trichiniasis. One patient died. Two months after burial the body was examined, and living and dead nematode worms were found which, as Bastian showed, were not the trichina, but a rhabditis.

They were probably not parasitic, but entered the body of the cadet after burial.

Prophylaxis.—It is not definitely known how swine become diseased. It has been thought that they are infected from rats about slaughter-houses, but it is just as reasonable to believe that the rats are infected by eating portions of the trichinous flesh of swine. The swine should, as far as possible, be grain-fed, and not, as is so common, allowed to eat offal. The most satisfactory prophylaxis is the complete cooking of pork and sausages, and to this custom in England, France, South Germany, and particularly in this country, immunity is largely due.

Treatment.—If it has been discovered within twenty-four or thirty-six hours that a large number of persons have eaten infected meat, the indications are to thoroughly evacuate the gastro-intestinal canal. Purgatives of rhubarb and senna may be given, or an occasional dose of calomel. Glycerin has been recommended in large doses in order that by passing into the intestines it may by its hygroscopic properties destroy the worm. Male-fern, kamala, santonin, and thymol have all been recommended in this stage. There is no doubt that diarrhœa in the first week or ten days of the infection is distinctly favorable. The indications in the stage of invasion are to relieve the pains, to secure sleep, and to support the patient's strength. There are no medicines which have any influence upon the embryos in their migration through the muscles.

III. Anchylostomiasis.

The *dochmius* or *strongylus duodenalis*, also known as the *sclerostomum* or *anchylostomum duodenale* is the only strongyle harmful to man. It belongs to the same family as the *strongylus armatus*, which causes the verminous aneurism in the horse. The parasites live in the upper portion of the small intestine, chiefly in the jejunum. They are easily seen, the male having a length of from six to ten millimetres, and the female from ten to eighteen millimetres. The mouth is provided with a series of tooth-like hooks, by means of which the parasite attaches itself to the mucous membrane. The male has a prominent expansion or bursa at the tail end. The existence of the parasite has long been known, but it was not thought to be pathogenic until Griesinger demonstrated its association with the Egyptian chlorosis. It has also been shown to be the cause of the anæmia to which miners and brick-makers are subject. Throughout Europe the disease has been widely spread by the employment of Italian and Polish laborers. In certain Italian provinces it is extremely prevalent and serious. It occurs in India and in Brazil, and has been described in Jamaica (Strachan). Dolley states that the parasite was described many years ago by physicians in the Southern States, but no recent observations upon the disease have been made in this country.

Symptoms.—The parasites withdraw blood by suction, and the

symptoms result from this slow depletion. In the early stage there may only be gastric or gastro-intestinal disturbance, but if the parasites are present in large numbers anæmia is gradually produced and constitutes the characteristic feature of the disease. The Egyptian chlorosis, brick-maker's anæmia, tunnel anæmia, miner's cachexia, and mountain anæmia are due to this cause. The clinical course is variable. In some instances the anæmia develops acutely and reaches a high grade within a short time, causing great shortness of breath and œdema. There is serious disturbance of nutrition, sometimes diarrhœa and colicky pains; but the most pronounced symptom is the pallor and the associated phenomena of chronic anæmia. The lesions of the intestines are those of chronic catarrh, and small hæmorrhages occur in the mucosa. Dilatation and hypertrophy of the heart have been found in many cases.

The diagnosis is not difficult. The ova, which are abundant in the stools, are oval, about ·05 millimetre in length, and possess a thin, transparent shell. There is no operculum, as in the ovum of the oxyuris, and the yolk is unsegmented. The larvæ develop in moist earth and readily get into the drinking water, through which infection occurs.

The systematic use of latrines and the boiling of all water used for drinking purposes are the important prophylactic measures. The treatment should be directed to the destruction of the parasites in the intestine, which may be effected by the male fern or by thymol, which Sonsino recommends highly. It is given in capsules of half a drachm every hour for four doses. A purgative is not necessary.

IV. Filariasis.

Under this term may be considered the morbid conditions induced by the *filaria sanguinis hominis*, or the *filaria Bancrofti*, the name employed to designate the adult worm, which was discovered by Bancroft, of Brisbane. In the adult form the worm lives in the lymphatics. The female is thus described by Patrick Manson, whose studies on this parasite have been so important: "A long, slender, hair-like animal quite three inches in length but only one one hundredth inch in breadth, of an opaline appearance, looking, as it lies in the tissues, like a delicate thread of catgut animated and wriggling. A narrow alimentary canal runs from the simple club-like head to within a short distance of the tail, the remainder of the body being almost entirely occupied by the reproductive organs. The vagina opens about one twenty-fifth of an inch from the head; it is very short, and bifurcates into two uterine horns, which, stuffed with embryos in all stages of development, run backward nearly to the tail." The male worm is much smaller and has only occasionally been found. The female produces an extraordinary number of embryos, which enter the blood current through the lymphatics. Each embryo is within its shell, which is elongated, scarcely perceptible, and in no way impedes the movements.

They are about the ninetieth part of an inch in length and the diameter of a red blood-corpuscle in thickness, so that they readily pass through the capillaries. They move with the greatest activity and form very striking and readily recognized objects in a blood-drop under the microscope. A remarkable feature is the periodicity in the occurrence of the embryos in the blood. In the daytime they are almost or entirely absent, whereas at night, in typical cases, they are present in large numbers. If, however, as Stephen Mackenzie has shown, the patient, reversing his habits, sleeps during the day, the periodicity is reversed. The further development of the embryos appears to be associated with the mosquito, which at night sucks the blood and in this way frees them from the body. Some slight development takes place within the body of the mosquito, and it is probable that the embryos are set free in the water after the death of the host. The further development is not known, but it is probably in drinking water. The filariæ may be present in the body without causing any symptoms. In animals blood filariæ are very common and rarely cause inconvenience. It is only when the adult worms or the ova block the lymph channels that certain definite symptoms occur. Manson suggests that it is the ova (prematurely discharged), which are considerably shorter and thicker than the full-grown embryos, which block the lymph channels and produce the conditions of hæmatochyluria, elephantiasis, and lymph-scrotum.

The parasite is widely distributed, particularly in tropical and subtropical countries. Guitéras has shown that the disease prevails extensively in the Southern States, and since his paper appeared contributions have been made by Matas, of New Orleans, Mastin, of Mobile, and De Saussure, of Charleston.

The effects produced may be described under the above-mentioned conditions.

(*a*) *Hæmatochyluria.*—Without any external manifestations, and in many cases without special disturbance of health, the subject from time to time passes urine of an opaque white, milky appearance, or bloody, or a chylous fluid which on settling shows a slightly reddish clot. The urine may be normal in quantity or increased. The condition is usually intermittent, and the patient may pass normal urine for weeks or months at a time. Microscopically, the chylous urine contains minute molecular fat granules, usually red blood-corpuscles in various amounts. It was in urine of this kind that Wucherer, of Bahia, first detected the filarian embryos. It is remarkable for how long the condition may persist without serious impairment of the health. A patient, sent to me by Dawson, of Charleston, has had hæmatochyluria intermittently for eighteen years. The only inconvenience has been in the passage of the blood-clots which collect in the bladder. At times he has also uneasy sensations in the lumbar region. The embryos are present in his blood at night in large numbers. Chyluria is not always due to the filaria. The non-parasitic form of the disease has already been considered.

Opportunities for studying the anatomical condition of these cases rarely occur. In the case described by Stephen Mackenzie the renal and peritoneal lymph plexuses were enormously enlarged, extending from the diaphragm to the pelvis. The thoracic duct above the diaphragm was impervious.

(*b*) *Lymph-scrotum* and certain forms of *elephantiasis* are sometimes caused by the filaria. In the former the tissues of the scrotum are enormously thickened and the distended lymph-vessels may be plainly seen. A clear, sometimes a turbid, fluid follows puncture of the skin. The parasites are not always to be found. I have examined two typical cases without finding filaria in the exuded fluids or in the blood at night. So also the majority of cases of elephantiasis which occur in this country are non-parasitic. In China it is stated that the parasites occur in all these cases.*

V. Dracontiasis (*Guinea-worm Disease*).

The *Filaria* or *Dracunculus medinensis* is a widely spread parasite in parts of Africa and the East Indies. In the United States cases occasionally occur. Jarvis reports a case in a post chaplain who had lived at Fortress Monroe, Va., for thirty years. Van Harlingen's patient, a man aged forty-seven, had never lived out of Philadelphia, so that the worm must be included among the parasites of this country. A majority of the cases reported in American journals have been imported.

Only the female is known. It develops in the subcutaneous and intermuscular connective tissues and produces vesicles and abscesses. In the large majority of the cases the parasite is found in the leg. Of 181 cases, in 124 the worm was found in the feet, 33 times in the leg, and 11 times in the thigh. The worm is usually solitary, though there are cases on record in which six or more have been present. It is cylindrical in form, about two millimetres in diameter, and from fifty to eighty centimetres in length.

The worm gains entrance to the system through the stomach, not through the skin, as was formerly supposed. It is probable that both male and female are ingested; but the former dies and is discharged, while the latter after impregnation penetrates the intestine and attains its full development in the subcutaneous tissues, where it may remain quiescent for a long time and can be felt beneath the skin like a bundle of string. Suppuration is after a time excited, and when the abscesses are opened or burst the worm appears and is sometimes discharged entire. The worm contains an enormous number of living embryos, which escape into the water and develop in the cyclops—a small crustacean—and it seems likely that man is infected by drinking the water containing these developed larvæ.

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* For full consideration of the subject of congenital occlusion and dilatation of lymph channels, see work on this subject by Samuel C. Busey, New York, 1878.

The *treatment* consists in promoting the suppuration, and when the worm is seen the common procedure is to roll it round a portion of smooth wood, and in this way prevent the retraction, and each day wind a little more until the entire worm is withdrawn. It is stated that special care must be taken to prevent tearing of the worm, as disastrous consequences sometimes follow, probably from the irritation caused by the migration of the embryos. It is stated that the leaves of the plant called *amarpattee* are almost a specific in the disease. Asafœtida in full doses is said to kill the worm.

VI. Other Nematodes.

(*a*) Among less important filarian worms parasitic in man the following may be mentioned: *filaria loa*, which is a cylindrical worm of about three centimetres in length and whose habitat is beneath the conjunctiva. It has been found on the West African coast, in Brazil, and in the West Indies. *Filaria lentis*, which has been found in a cataract. Three specimens have been found together. *Filaria labialis*, which has been found in a pustule in the upper lip *Filaria hominis oris*, which was described by Leidy, from the mouth of a child. *Filaria bronchialis*, which has been found occasionally in the trachea and bronchi. This parasite has been seen in a few cases in the bronchioles and in the lungs. There is no evidence that it ever produces an extensive verminous bronchitis similar to that which I have described in dogs. *Filaria imitis*, of which Bowlby has described two cases. In one case with hæmaturia female worms were found in the portal vein, and the ova were present in the thickened bladder wall and in the ureters.

(*b*) *Trichocephalus Dispar* (*Whip-worm*).—This parasite is not infrequently found in the cæcum and large intestine of man. It measures from four to five centimetres in length, the male being somewhat shorter than the female The worm is readily recognized by the remarkable difference between the anterior and posterior portions. The former, which is at least three fifths of the body, is extremely thin and hair-like in contrast to the thick hinder portion of the body, which in the female is conical and pointed, and in the male more obtuse and usually rolled like a spring. The ova are, oval, lemon-shaped, ·05 millimetre in length, and each is provided with a button-like projection.

The number of the worms found is variable, as many as a thousand having been counted. It is a widely spread parasite. In parts of Europe it occurs in from ten to thirty per cent of all bodies examined, but in this country it is not so common. The trichocephalus rarely causes symptoms. It has been thought by certain physicians in the East to be the cause of beri-beri Several cases have been reported recently in which profound anæmia has occurred in connection with this parasite, usually with diarrhœa. Enormous numbers may occur, as in Rudolphi's case, without producing any symptoms.

The diagnosis is readily made by the examination of the fæces, which contain, sometimes in great abundance, the characteristic lemon-shaped, hard, dark-brown eggs.

(*c*) *Eustrongylus Gigas.*—This enormous nematode, the male of which measures about a foot in length and the female about three feet, occurs in very many animals and has occasionally been met with in man. It is usually found in the renal region and may entirely destroy the kidney.

(*d*) *Rhabdonema Intestinale.*—Under this name are now included the small nematode worms found in the fæces and formerly described as *anguillula stercoralis* and *anguillula intestinalis.* This parasite occurs abundantly in the stools of the endemic diarrhœa of hot countries, and has been specially described by the French in the diarrhœa of Cochin-China. It occurs also in Brazil, and has been found in Italy in connection with the anchylostoma in cases of miner's anæmia. It is stated that the worms occupy all parts of the intestines, and have even been found in the biliary and pancreatic ducts. It is only when they are in very large numbers that they produce severe diarrhœa and anæmia.

Acanthocephala (*Thorn-headed Worms*).—The *echinorhynchus gigas* is a common parasite in the intestine of the hog and attains a large size. The larvæ develop in cockchafer grubs. Lambl found a small *echinorhynchus* in the intestine of a boy. Welch's specimen, which was found encysted in the intestine of a soldier at Netley, is stated by Cobbold probably not to have been an *echinorhynchus.* Recently a case of *echinorhynchus moniliformis* has been described in Italy by Grassi and Calandruccio.

IV. DISEASES CAUSED BY CESTODES

(*Tape-worms; Hydatid Disease*).

Man harbors the adult parasites in the small intestine, the larval forms in the muscles and solid organs.

I. Intestinal Cestodes; Tape-worms.

(*a*) *Tænia solium*, or pork tape-worm. This is not a common form in this country. It is much more frequent in parts of Europe and Asia. When mature it is from six to twelve feet in length. The head is small, round, not so large as the head of a pin, and provided with four sucking disks and a double row of hooklets; hence it is called, in contradistinction to the other form in man, the armed tape-worm. To the head succeeds a narrow, thread-like neck, then the segments, or proglottides, as they are called. The segments possess both male and female generative organs, and about the four hundred and fiftieth become mature and contain ripe ova. The worm attains its full growth in from three to three and a half months, after which time the segments are continuously shed and appear

in the stools. The segments are about one centimetre in length and from seven to eight millimetres in breadth. Pressed between glass plates the ovarian rosette is seen as a central stem with about twelve or fifteen lateral branches. There are many thousands of ova in each ripe segment, and each ovum consists of a firm shell, inside of which is a little embryo, provided with six hooklets. The segments are continuously passed, and if the ova are to attain further development they must be taken into the stomach, either of a pig, or of man himself. The egg-shells are digested, the six-hooked embryos become free, and passing from the stomach reach various parts of the body (the liver, muscles, brain, or eye), where they develop into the larvæ or cysticerci. A hog under these circumstances is said to be *measled*, and the cysticerci are spoken of as measles or bladder worms.

The *tænia solium* received its name because it was thought to exist as a solitary parasite in the bowel, but two or three, or even more worms may occur.

(*b*) *Tænia saginata* or *mediocanellata*—the unarmed or beef tape-worm. This is a longer and larger parasite than the *tænia solium*. It is certainly the common tape-worm of this country. Of scores of specimens which I have examined, almost all were of this variety. According to Bérenger-Féraud it has spread rapidly in western Europe, owing probably to the importation of beef and live stock from the Mediterranean basin. It may attain a length of fifteen or twenty feet, or more. The head is large in comparison to the *tænia solium*, and measures over two millimetres in breadth. It is square-shaped and provided with four large sucking disks, but there are no hooklets. The ripe segments are from seventeen to eighteen millimetres in length, and from eight to ten millimetres in breadth. The ovarian rosette consists of a central stem with from seventeen to eighteen lateral branches, which are given off more dichotomously than in the *tænia solium*. The ova are somewhat larger, and the shell is thicker, but the two forms can scarcely be distinguished by their ova. The ripe segments are passed as in the tænia solium, and are ingested by cattle, in the flesh or organs of which the eggs develop into the bladder worms or cysticerci. Whether they develop in man or not is uncertain. No instance of the cysticercus of the *tænia saginata* has, so far as I know, been reported in man.

Of other forms of tape-worm may be mentioned:

(*c*) *Tænia elliptica* (*tænia cucumerina*). A small parasite very common in the dog and occasionally found in man, and the larvæ of which develop in the louse of the dog.

(*d*) *Tænia flavo-punctata.* A small cestode was found in the intestine of a child in Boston, and has since been met with in one or two cases.

(*e*) *Tænia nana* and the *tænia Madagascariensis* have been found only once or twice.

(*f*) *Bothriocephalus latus.* A cestode worm found only in certain districts bordering on the Baltic Sea and in parts of Switzerland. So far as I know it has not been found in this country except in a few imported cases. The parasite is large and long, measuring from twenty-five to thirty feet or more. Its head is different from that of the tænia, as it possesses two lateral grooves or pits and has no hooklets. The larvæ develop in the peritonæum and muscles of the pike and other fish, and it has been shown experimentally that they grow into the adult worm when eaten by man.

Symptoms.—These parasites are found at all ages. They are not uncommon in children and are occasionally found in sucklings. W. T. Plant refers to a number of cases in children under two years, and there is a case in the literature in which it is stated that the tape-worm was found in an infant five days old.

The parasites may cause no disturbance and are rarely dangerous. A knowledge of the existence of the worm is generally a source of worry and anxiety; the patient may have considerable distress and complain of abdominal pains, nausea, and sometimes diarrhœa. Occasionally, the appetite is ravenous. In women and in nervous patients the constitutional disturbance may be considerable, and we not infrequently see great mental depression and even hypochondria. Various nervous phenomena, such as chorea, convulsions, or epilepsy, are believed to be caused by the parasites. Such effects, however, are very rare.

The *diagnosis* is never doubtful. The presence of the segments is distinctive. The ova, too, may be recognized in the stools. It makes but little difference as to the form of tape-worm, but the ripe segments of the *tænia saginata* are larger and broader, and show differences in the generative system as already mentioned.

The *prophylaxis* is most important. Careful attention should be given to two points. First, all tape-worm segments should be burned. They should never be thrown into the water-closet or outside. And second, the meat should be cooked throughout, in which way alone larvæ are destroyed. Possibly it is owing to the fact that in this country pork is, as a rule, better cooked than beef that the *tænia saginata* is the most common form. Certainly in the market and at the abattoirs one more commonly sees measly pork than measly veal. In the examination of a thousand hogs in Montreal there were seventy-six instances of cysticerci. The measle is more readily overlooked in beef than in pork, as in the former it has not such an opaque white color.

Treatment.—For two days prior to the administration of the remedies the patient should take a very light diet and have the bowels moved occasionally by a saline cathartic. The practitioner has the choice of a large number of drugs. As a rule, the male fern acts promptly and well. The ethereal extract, in two-drachm doses, may be given fasting, and followed in the course of a couple of hours by a brisk purgative. This usu-

ally succeeds in bringing away a large portion, but not always the entire worm.

A combination of the remedies is sometimes very effective. An infusion is made of pomegranate root, half an ounce; pumpkin seeds, one ounce; powdered ergot, a drachm; and boiling water, ten ounces. To an emulsion of the male fern (a drachm of ethereal extract), made with acacia powder, two minims of croton oil are added. The patient should have had a low diet the previous day and have taken a dose of salts in the evening. The emulsion and infusion are mixed and taken fasting at nine in the morning.

The pomegranate root is a very efficient remedy, and may be given as an infusion of the bark, three ounces of which may be macerated in ten ounces of water and then reduced to one half by evaporation. The entire quantity is then taken in divided doses. It occasionally produces colic, but is a very effective remedy. The active principle of the root, pelletierine, is now much employed. It is given in doses of one fourth to one half of a grain, and is followed in an hour by a purge.

Pumpkin seeds are sometimes very efficient. Three or four ounces should be carefully bruised and then macerated for twelve or fourteen hours and the entire quantity taken and followed in an hour by a purge. Of other remedies, koosso, turpentine in ounce doses in honey, and kamala may be mentioned.

Unless the head is brought away, the parasite continues to grow, and within a few months the segments again appear. Some instances are extraordinarily obstinate. Doubtless it depends a good deal upon the exposure of the worm. The head and neck may be thoroughly protected beneath the valvulæ conniventes, in which case the remedies may not act. Owing to its armature the *tænia solium* is more difficult to expel. It is probable that no degree of peristalsis could dislodge the head, and unless the worm is killed it does not let go its extraordinarily firm hold on the mucous membrane.

II. Visceral Cestodes.

Whereas adult tæniæ cause little or no disturbance and rarely, if ever, prove directly fatal, the affections caused by the larvæ or immature forms in the solid organs are serious and important. There are two chief cestode larvæ known to frequent man—(*a*) the *cysticercus cellulosæ*, the larva of the *tænia solium*, and (*b*) the *echinococcus*, the larva of the *tænia echinococcus*.

I. **Cysticercus Cellulosæ.**—When man accidentally takes into his stomach the ripe ova of *tænia solium* he is liable to become the intermediate host, a part usually played for this tape-worm by the pig. This accident may occur in an individual the subject of *tænia solium*, in which case the mature proglottides either themselves wander into the stomach or, what is more likely, are forced into the organ in attacks of prolonged

vomiting. Of course the accidental ingestion from the outside of a few ova is quite possible, and the liability of infection should always be borne in mind in handling the segments of the worm.

The symptoms depend entirely upon the number of ova ingested and the localities reached. In the hog the cysticerci produce very little disturbance. The muscles, the connective tissue, and the brain may be swarming with the measles, as they are called, and yet the nutrition is maintained and the animal does not appear to be seriously incommoded. In the invasion period, if large numbers of the parasites are taken, there is, in all probability, constitutional disturbance; certainly there is in the calf, when fed with the ripe segments of *tænia saginata*.

In man a few cysticerci lodged beneath the skin or in the muscles may cause no damage, and in time the larvæ die and become calcified. They are occasionally found in dissection subjects or in post-mortems as ovoid white bodies in the muscles or subcutaneous tissue. In this country they are very rare. I have seen but one instance in my post-mortem experience. Depending on the number and the locality specially affected, the symptoms may be grouped into general, cerebro-spinal, and ocular.

(1) *General.*—As a rule the invasion of the larvæ in man, unless in very large numbers, does not cause very definite symptoms. It occasionally happens, however, that a striking picture is produced. For instance, a patient was admitted to my wards very stiff and helpless, so much so that he had to be assisted up-stairs and into bed. He complained of numbness and tingling in the extremities and general weakness, so that at first he was thought to have a peripheral neuritis. At the examination, however, a number of painful subcutaneous nodules were discovered, which proved on excision to be the cysticerci. Altogether seventy-five could be felt subcutaneously, and from the soreness and stiffness they probably existed in large numbers in the muscles. There were none in his eyes, and he had no symptoms pointing to brain lesions.

(2) *Cerebro-spinal.*—Remarkable symptoms may result from the presence of the cysticerci in the brain and cord. In the silent region they may be abundant without producing any symptoms. I have in my possession the brain of a pig containing scores of "measles," yet the animal in the few moments in which I saw it just prior to death did not present any symptoms to attract attention. In the ventricles of the brain the cysticerci may attain a considerable size, owing to the fact that in regions in which they are unrestrained in their growth the bladder-like body grows freely, as in the peritonæum. When in the fourth ventricle remarkable irritative symptoms may be produced. In 1884 I saw with Friedländer in Berlin a case from Riess's wards in which during life there had been symptoms of diabetes and anomalous nervous symptoms. Post mortem, the cysticercus was found beneath the valve of Vieussens, pressing upon the floor of the left ventricle.

(3) *Ocular.*—Since von Graefe demonstrated the presence of the cysti-

cercus in the vitreous humor many cases have been placed on record, and it is a condition easily recognized by oculists.

Except in the eye, the diagnosis can rarely be made; when the cysticerci are subcutaneous, one may be excised. It is possible that when numerous throughout the muscles they may be seen under the tongue, in which situation they may exist in the pig in numbers.

II. **Echinococcus Disease.**—The hydatid worms or echinococci are the larvæ of the *tænia echinococcus* of the dog. This is a tiny cestode not more than four or five millimetres in length, consisting of only three or four segments, of which the terminal one alone is mature, and has a length of about two millimetres and a breadth of 0·6 millimetre. The head is small and provided with four sucking disks and a rostellum with a double row of hooklets. This is an exceedingly rare parasite in the dog. Cobbold states that he has never met with a natural specimen in England. Leidy had not one in his large collection. I have not met with an instance in this country, nor do I know of its ever having been described. The only specimens in my cabinet I procured experimentally by feeding a dog with echinococcus cysts from an ox. The worms are so small that they may be readily overlooked, since they form small white, thread-like bodies closely adherent among the villi of the small intestines. The ripe segment contains about 5,000 eggs, which attain their development in the solid organs of various animals, particularly the hog and ox; more rarely the horse and the sheep. In some countries man is a common intermediate host, owing to the accidental ingestion of the ova.

Development.—The little six-hooked embryo, freed from the egg-shell by digestion, either burrows through the intestinal wall and reaches the peritoneal cavity or the muscles; more commonly it enters the portal vessels and is carried to the liver. It may enter the systemic vessels, and, passing the pulmonary capillaries, as it is protoplasmic and elastic, may reach the brain or other parts. Once having reached its destination, it undergoes the following changes: The hooklets disappear and the little embryo is gradually converted into a small cyst which presents two distinct layers—an external, laminated, cuticular membrane or capsule, and an internal, granular, parenchymatous layer, the endocyst. The little cyst or vesicle contains a clear fluid. There is more or less reaction in the neighboring tissues, and the cyst in time has a fibrous investment. When this primary cyst or vesicle has attained a certain size buds develop from the parenchymatous layer, which are gradually converted into cysts, presenting a structure identical with that of the original cyst, namely, an elastic chitinous membrane lined with a granular parenchymatous layer. These secondary or daughter cysts are first connected with the lining membrane of the primary, but are soon set free. In this way the primary cyst as it grows may contain a dozen or more daughter cysts. Inside these daughter cysts a similar process may occur, and from buds in the walls granddaughter cysts are developed. From the granular layer of the parent and

daughter cysts buds arise which develop into brood capsules. From the lining membrane the little outgrowths arise and gradually develop into bodies known as scolices, which represent in reality the head of the *tænia echinococcus* and present four sucking disks and a circle of hooklets. Each scolex is capable when transferred to the intestines of a dog of developing into an adult tape-worm. The difference between the ovum of an ordinary tape-worm, such as the *tænia solium*, and the *tænia echinococcus* is in this way very striking. In the former case the ovum develops into a single larva—the *cysticercus cellulosæ*—whereas the egg of the *tænia echinococcus* develops into a cyst which is capable of multiplying enormously and from the lining membrane of which millions of larval tape-worms develop. Ordinarily in man the development of the echinococcus takes place as above mentioned and by an endogenous form in which the secondary and tertiary cysts are contained within the primary; but in animals the formation may be different, as the buds from the primary cyst penetrate between the layers and develop externally, forming the *exogenous* variety. A third form is the multilocular echinococcus, in which from the primary cysts buds develop which are cut off completely and are surrounded by thick capsules of a connective tissue, which join together and ultimately form a hard mass represented by strands of connective tissue enclosing alveolar spaces about the size of peas or a little larger. In these spaces are found the remnants of the echinococcus cyst, occasionally the scolices or hooklets, but they are often sterile.

The fluid of the echinococcus cysts is clear and limpid, and has a specific gravity from 1·005 to 1·009. It does not contain albumen, but may contain traces of sugar. As a rule, the cysts, when not degenerated, contain the hydatid heads or scolices or the characteristic hooklets.

Changes in the Cyst.—It is not known definitely how long the echinococcus remains alive, but it probably lives many years—according to some authors as long as twenty years. The most common change is death and the gradual inspissation of the contents and conversion of the cyst into a mass containing putty-like or granular material which may be partially calcified. Remnants of the chitinous cyst wall or hooklets may be found. These obsolete hydatid cysts are not infrequently found in the liver. A more serious termination is rupture, which may take place into a serous sac, or perforation may take place externally, when the cysts are discharged, as into the bronchi or alimentary canal or urinary passages. More unfavorable are the instances in which rupture occurs into the bile-passages or into the inferior cava. Recovery may follow the rupture and discharge of the hydatids externally. Sudden death has been known to follow the rupture. A third and very serious mode of termination is suppuration, which may occur spontaneously or follow rupture and is found most frequently in the liver. Large abscesses may be formed which contain the hydatid membranes.

Geographical Distribution of the Echinococcus.—The disease prevails

most extensively in those countries in which man is brought into close contact with the dog, particularly when, as in Australia, the dogs are used extensively for herding sheep, the animal in which the larval form of the *tænia echinococcus* is most frequently found. In Iceland the cases are very numerous. In Europe the disease is not uncommon. In this country it is extremely rare and a great majority of all cases are in foreigners. Up to July, 1891, I have been able to find in the literature (and in the museums) only 85 cases in the United States and Canada.*

Distribution in the Body.—Of the 1,862 cases comprised in the statistics of Davaine, Cobbold, Finsen, and Neisser, the parasites existed in the liver in 953, in the intestinal canal in 163, in the lung or pleura in 153, in the kidneys, bladder, and genitals in 186, in the brain and spinal canal in 127, bone 61, heart and blood-vessels 61, other organs 158.† Of the 85 cases in this country, the liver was the seat of the disease in 59. Of 50 consecutive cases treated by Mosler at the Greifswald clinic, 36 involved the liver, 10 the lungs, 3 the right kidney, and 1 the spleen.

Symptoms.—(*a*) *Hydatids of the Liver.*—Small cysts may cause no disturbance; large and growing cysts produce signs of tumor of the liver with great increase in the size of the organ. Naturally the physical signs depend much upon the situation of the growth. Near the anterior surface in the epigastric region the tumor may form a distinct prominence and have a tense, firm feeling, sometimes with fluctuation. A not infrequent situation is to the left of the suspensory ligament, forming a tumor which pushes up the heart and causes an extensive area of dulness in the lower sternal and left hypochrondriac regions. In the right lobe, if the tumor is on the posterior surface, the enlargement of the organ is chiefly upward into the pleura and the vertical area of dulness in the posterior axillary line is increased. Superficial cysts may give what is known as the hydatid fremitus. If the tumor is palpated lightly with the fingers of the left hand and percussed at the same time with those of the right there is felt a vibration or trembling movement which persists for a certain time. It is not always present, and it is doubtful whether it is peculiar to the hydatid tumors or is due, as Briançon held, to the collision of the daughter cysts. Very large cysts are accompanied by feelings of pressure or dragging in the hepatic region, sometimes actual pain. The general condition of the patient is at first good and the nutrition little, if at all, interfered with. Unless some of the accidents already referred to occur, the symptoms indeed may be trifling and due only to the pressure or weight of the tumor.

Suppuration of the cyst changes the clinical picture into one of pyæmia. There are rigors, sweats, more or less jaundice, and rapid loss of weight. Perforation may occur into the stomach, colon, pleura, bronchi, or exter-

* American Journal of the Medical Sciences, October, 1882. Since that date Alfred Mann has collected for me 24 cases in addition to the 61 there reported.

† Dictionnaire Encyclopédique des Sciences Médicales, tome 32, 1885.

nally, and in some instances recovery has taken place. Perforation into the pericardium and inferior vena cava is fatal. In the latter case the daughter cysts have been found in the heart, plugging the tricuspid orifice and the pulmonary artery. Perforation of the bile-passages causes intense jaundice, and may lead to suppurative cholangitis.

An interesting symptom connected with the rupture of hydatid cysts is the development of urticaria, which may also follow aspiration of the cysts and is probably due to the absorption of toxic materials contained in the fluid.

Diagnosis. — Cysts of moderate size may exist without producing symptoms. Large multiple echinococci may cause great enlargement with irregularity of the outline, and such a condition persisting for any time with retention of the health and strength suggests hydatid disease. An irregular, painless enlargement, particularly in the left lobe, or the presence of a large, smooth fluctuating tumor of the epigastric region is also very suggestive, and in this situation, when accessible to palpation, it gives a sensation of a smooth elastic growth and possibly also the hydatid tremor. When suppuration occurs the clinical picture is really that of abscess and only the existence of previous enlargement of the liver with good health would point to the fact that the suppuration was associated with hydatids. Syphilis may produce irregular enlargement without much disturbance in the health, sometimes also a very definite tumor in the epigastric region, but it is usually firm and not fluctuating. The clinical features may simulate cancer very closely. In a case which I reported the liver was greatly enlarged and there were many nodular tumors in the abdomen. The post-mortem showed enormous suppurating hydatid cysts in the left lobe of the liver which had perforated the stomach in two places and also the duodenum. The omentum, mesentery, and pelvis also contained numerous cysts. As a rule, the clinical course of the disease would suffice to separate it clearly from cancer. Dilatation of the gall-bladder and hydronephrosis have both been mistaken for hydatid disease. In the former the movable character of the tumor, its shape, and the mucoid character of the contents suffice for the diagnosis. In some instances of hydronephrosis only the exploratory puncture could distinguish between the conditions. More frequent is the mistake of confounding a hydatid cyst of the right lobe pushing up the pleura with pleural effusion of the right side. The heart may be dislocated, the liver depressed, and dulness, feeble breathing, and diminished fremitus are present in both conditions. Frerichs lays stress upon the different character of the line of dulness; in the echinococcus cyst the upper limit presents a curved line, the maximum of which is usually in the scapular region. Suppurative pleurisy may be caused by the perforation of the cyst. If adhesions result, the perforation takes place into the lung, and fragments of the cysts or small daughter cysts may be coughed up. For diagnostic purposes the exploratory puncture should be used. As

stated, the fluid is usually perfectly clear or slightly opalescent, the reaction is neutral, and the specific gravity varies from 1·005 to 1·009. It is non-albuminous, but contains chlorides and sometimes traces of sugar. Hooklets may be found either in the clear fluid or in the suppurating cysts. They are sometimes absent, however, as the cyst may be sterile.

(*b*) *Echinococcus of the Respiratory System.*—The larvæ may develop primarily in the pleura and attain a large size. The symptoms are at first those of compression of the lung and dislocation of the heart. The physical signs are those of fluid in the pleura and the condition could scarcely be distinguished from ordinary effusion. The line of dulness may be quite irregular. As in the echinococcus of the liver, the general condition of the patient may be excellent in spite of the existence of extensive disease. Pleurisy is rarely excited. The cysts may become inflamed and perforate the chest wall. In a case of D. F. Smith's, of Walkertown, Ontario, a girl, aged twenty, had a running sore in the eighth left intercostal space. This was freely opened, and in the pus which flowed out were a number of well-characterized echinococcus cysts of various size. The patient recovered.

Echinococci occur more frequently in the lung than in the pleura. If small, they may exist for some time without causing serious symptoms. In their growth they compress the lung and sooner or later lead to inflammatory processes, often to gangrene, and the formation of cavities which connect with the bronchi. Fragments of membrane or small cysts may be expectorated. Hæmorrhage is not infrequent. Perforation into the pleura with empyema is common. A majority of the cases are regarded during life as either phthisis or gangrene, and it is only the detection of the characteristic membranes or the hooklets which leads to the diagnosis. The condition is usually fatal; only a few cases have recovered. Of the 85 American cases, in six the cysts occurred in the lung or pleura.

(*c*) *Echinococcus of the Kidneys.*—In the collected statistics referred to above the genito-urinary system comes second as the seat of hydatid disease, though it is rare in comparison with the affection of the liver. Of the 85 American cases, there were only three in which the kidneys or bladder were involved. The kidney may be converted into an enormous cyst resembling hydronephrosis.

The diagnosis is only possible by puncture and examination of the fluid. The cyst may perforate into the pelvis of the kidney and portions of the membrane or cysts may be discharged with the urine, sometimes producing renal colic. I have reported a case in which for many months the patient passed at intervals numbers of small cysts with the urine. The general health was little if at all disturbed, except by the attacks of colic during the passage of the parasites.

(*d*) *Echinococcus of the Nervous System.*—In this country very few instances have occurred in the brain. One or two reports indicate clearly that the common cystic disease of the choroidal plexuses has been mis-

taken for hydatids. Davies Thomas, of Australia, has tabulated 97 cases, including some of the *cysticercus cellulosæ*. According to his statistics, the cyst is more common on the right than on the left side, and is most frequent in the cerebrum.

The symptoms are very indefinite, as a rule, being those of tumor. Persistent headache, convulsions, either limited or general, and gradually developing blindness have been prominent features in many cases.

Multilocular Echinococcus.—This form merits a brief separate description, as it differs so remarkably from the usual type of the disease. About one hundred instances are on record, the great majority of which have occurred in Bavaria and in Switzerland. Only one case has been reported in the United States.* The patient was a German, who had been in the country five years. For a year previous to his death he was out of health, jaundiced, and somewhat emaciated. A fluctuating tumor was found in the right lumbar and umbilical regions, apparently connected with the liver. This was opened, and death followed from hæmorrhage. About a fourth of the right lobe of the liver was occupied by an irregular cavity with rough, ragged walls, which in places were from one to two inches in thickness and enclosed irregular small cavities. The lamellated cuticula characteristic of the echinococcus cyst was found lining these cavities. In some instances the tumor bears a striking likeness to colloid cancer, as on section it presents a fibrous stroma with cavities containing gelatinous material. They are often sterile—that is, without the hydatid heads or larvæ. This form is almost exclusively confined to the liver, and the symptoms resemble more those of tumor or cirrhosis. The liver is, as a rule, enlarged and smooth, not irregular as in the ordinary echinococcus. Jaundice is a common symptom. The spleen is usually enlarged, there is progressive emaciation, and toward the close hæmorrhages are common.

Treatment of Echinococcus Disease.—Medicines are of no avail. Post-mortem reports show that in a considerable number of cases the parasite dies and the cyst becomes harmless. Operative measures should be resorted to when the cyst is large or troublesome. The simple aspiration of the contents has been successful in a large number of cases, and as it is not in any way dangerous, it may be tried before the more radical procedure of incision and evacuation of the cysts. Suppuration has occasionally followed the puncture. Injections into the sac should not be practised. With modern methods surgeons now open and evacuate the echinococcus cysts with great boldness, and the Australian records, which are the most numerous and important on this subject, show that recovery is the rule in a large proportion of the cases. Suppurative cysts in the liver should be treated as abscess. Naturally the outlook is less favorable. The practical treatment of hydatid disease has been greatly

* Delafield and Prudden, Pathological Anatomy, third edition, page 317.

advanced by Australian surgeons. The recent work of James Graham, of Sydney, may be consulted for interesting details in diagnosis and treatment.

V. PARASITIC ARACHNIDA.

(1) **Pentastomes.**—(*a*) The *pentastomum tænioides* has a somewhat lancet-shaped body, the female from three to four inches in length, the male about an inch in length. The body is tapering and marked by numerous rings. The adult worm infests the frontal sinuses and nostrils of the dog, more rarely of the horse. The larval form, which is known as the *pentastomum denticulatum*, is found in the internal organs, particularly the liver, but has also been found in the kidney. The adult worm has been found in the nostril of man, but is very rare and seldom occasions any inconvenience. The larvæ are by no means uncommon, particularly in parts of Germany.

(*b*) The *pentastomum constrictum*, which is about the length of half an inch, with twenty-three rings on the abdomen, was found by Aitken in the liver and lungs of a soldier of a West Indian regiment.

The only case of pentastomes which, so far as I know, has been reported in this country is the one referred to in Flint's Practice of Medicine. From 75 to 100 of the parasites were expectorated. The liver was enlarged and the parasites probably occupied this region. In 1869 I saw a specimen which had been passed in the urine by a patient of James H. Richardson, of Toronto.

(2) **Demodex (Acarus) Folliculorum.**—A minute parasite, from 0·3 millimetre to 0·4 millimetre in length, which lives in the sebaceous follicles, particularly of the face. It is doubtful whether it produces any symptoms. Possibly when in large numbers they may excite inflammation of the follicles, leading to acne.

(3) **Acarus (or Sarcoptes) Scabiei** (*Itch Insect*).—This is the most important of the arachnid parasites, as it produces troublesome and distressing skin eruptions. The male is ·23 millimetre in length, and ·19 millimetre in breadth; the female is 0·45 millimetre in length and 0·35 millimetre in width. The female can be seen readily with the naked eye and has a pearly-white color. It is not so common a parasite in the United States and Canada as in Europe.

The insect lives in a small burrow, about one centimetre in length, which it makes for itself in the epidermis. At the end of this burrow the female lives. The male is seldom found. The chief seat of the parasite is in the folds where the skin is most delicate, as in the web between the fingers and toes, the backs of the hands, the axilla, and the front of the abdomen. The head and face are rarely involved. The lesions which result from the presence of the itch insect are very numerous and result largely from the irritation of the scratching. The commonest is a papular

and vesicular rash or, in children, an ecthymatous eruption. The irritation and pustulation which follow the scratching may completely destroy the burrows, but in typical cases there is rarely any doubt as to the diagnosis.

The treatment is simple. It should consist of warm baths with a thorough use of a soft soap, after which the skin should be anointed with sulphur ointment, which in the case of children should be diluted. An ointment of naphthol (drachm to the ounce) is very efficacious.

(4) **Leptus Autumnalis** (*Harvest Bug*).—This reddish-colored parasite, about one half millimetre in size, is often found in large numbers in fields and in gardens. They attach themselves to animals and man with their sharp proboscides, and the hooklets of their legs produce a great deal of irritation. They are most frequently found on the legs. They are readily destroyed by sulphur ointment or corrosive-sublimate lotions.

Several varieties of ticks are occasionally found on man—the *Ixodes ricinus* and the *Ixodes americanus*, which are met with in horses and oxen.

VI. PARASITIC INSECTS.

(1) **Pediculi** (*Phthiriasis; Pediculosis*).—There are three varieties of the body louse, which are found only in persons of uncleanly habits.

Pediculus Capitis.—The male is from 1 to 1·5 millimetre in length and the female nearly 2 millimetres in length. The color varies somewhat with the different races of men. It is light gray with a black margin in the European, and very much darker in the negro and Chinese. They are oviparous, and the female lays about sixty eggs, which mature in a week. The ova are attached to the hairs, and can be readily seen as white specks, known popularly as nits. The symptoms are irritation and itching of the scalp. When numerous they may excite an eczema or a pustular dermatitis, which causes crusts and scabs, particularly at the back of the head. In the most extreme cases the hair becomes tangled in these crusts and matted together, forming at the occiput a firm mass which is known as *plica polonica*, as it was not infrequent among the Jewish inhabitants of Poland.

Pediculus Corporis (*Vestimentorum*).—This is considerably larger than the head louse. It lives on the clothing and in sucking the blood causes minute hæmorrhagic specks, which are very common about the neck, back, and abdomen. The irritation of the bites may cause urticaria, and the scratching is usually in linear lines. In long-standing cases, particularly in the old dissipated characters, the skin becomes rough and greatly pigmented, a condition which has been termed the vagabond's disease—*morbus errorum*—and which may be mistaken for the bronzing of Addison's disease.

Pediculus pubis differs somewhat from the other forms, and is found

in the parts of the body covered with short hairs, as the pubes; more rarely the axilla and eyebrows.

The *taches bleuâtres* are stated by French writers to be excited by the irritation of pediculi. They are certainly associated with them in a considerable number of cases, but, if really caused by these parasites, it is difficult to understand why they should only be present with fever.

Treatment.—For the *pediculus capitis*, when the condition is very bad, the hair should be cut short, as it is very difficult to destroy thoroughly all the nits. Repeated saturations of the hair in coal oil or in turpentine are usually efficacious, or with lotions of carbolic acid, one to fifty. Scrupulous cleanliness and care are sufficient to prevent recurrence. In the case of the *pediculus corporis* the clothing should be placed for several hours in a disinfecting oven. To allay the itching a warm bath containing four or five ounces of bicarbonate of soda is useful. The skin may be rubbed with a lotion of carbolic acid, two drachms to the pint, with two ounces of glycerin. For the *pediculus pubis* white precipitate or ordinary mercurial ointment should be used, and the parts should be thoroughly washed two or three times a day with soft soap and water.

(2) **Cimex Lectuarius** (*Common Bed-bug*).—This parasite is from three to four millimetres in length and has a reddish-brown color. It lives in the crevices of the bedstead and in the cracks in the floor and in the walls. It is nocturnal in its habits. The peculiar odor of the insect is caused by the secretion of a special gland. The parasite possesses a long proboscis, with which it sucks the blood. Individuals differ remarkably in the reaction to the bite of this insect; some are not disturbed in the slightest by them, in others the irritation causes hyperæmia and often intense urticaria. Thorough fumigation with sulphur or scouring with corrosive-sublimate solution destroys them.

(3) **Pulex Irritans** (*The Common Flea*).—The male is from 2 to 2·5 millimetres in length, the female from 3 to 4 millimetres. The flea is a transient parasite on man. The bite causes a circular red spot of hyperæmia in the centre of which is a little speck where the boring apparatus has entered. The amount of irritation caused by the bite is variable. Many persons suffer intensely and a diffuse erythema or an irritable urticaria develops; others suffer no inconvenience whatever.

The *pulex penetrans* (*sand-flea; jigger*) is found in tropical countries, particularly in the West Indies and South America. It is much smaller than the common flea, and not only penetrates the skin, but burrows and produces an inflammation with pustular or vesicular swelling. It most frequently attacks the feet. It is readily removed with a needle. Where they exist in large numbers the essential oils are used on the feet as a preventive.

VII. PSEUDO-PARASITES (*Myiasis*).

Of these, the most important are the larvæ of certain diptera, particularly the flesh flies—*creophila*. The condition is called myiasis.

The most common form is that in which an external wound becomes *living*, as it is called. This myiasis vulnerum is caused by the larvæ of either the blue-bottle or the common flesh fly. The larvæ can be removed readily with the forceps; if there is any difficulty, thorough cleansing and the application of an antiseptic bandage is sufficient to kill them. The ova of these flies may be deposited in the nostrils, the ears, or the conjunctiva—the myiasis narium, aurium, conjunctivæ. This invasion rarely takes place unless these regions are the seat of disease. In the nose and in the ear the larvæ may cause serious inflammation.

The cutaneous myiasis may be caused by the larvæ of the *musca vomitoria*, but more commonly by the bot-flies of the ox and sheep, which occasionally attack man. This condition is rare in temperate climates. Matas has described a case in which œstrus larvæ were found in the gluteal region. In parts of Central America the eggs of another bot-fly, the *dermatobia*, are not infrequently deposited in the skin and produce a swelling very like the ordinary boil.

Myiasis interna may result from the swallowing of the larvæ of the common house fly or of species of the genus *anthomyia*. There are many cases on record in which the larvæ of the *musca domestica* have been discharged by vomiting. Instances in which dipterous larvæ have been passed in the fæces are less common. Finlayson, of Glasgow, has recently reported an interesting case in a physician, who, after protracted constipation and pain in the back and sides, passed large numbers of the larvæ of the flower fly—*anthomyia canicularis*. Among other forms of larvæ or *gentles*, as they are sometimes called, which have been found in the fæces are those of the common house fly, the blue-bottle fly, and the *techomyza fusca*. The larvæ of other insects are extremely rare. It is stated that the caterpillar of the tabby moth has been found in the fæces.

Here may be mentioned among the effects of insects the remarkable *urticaria epidemica*, which is caused in some districts by the procession caterpillars, particularly the species *cnethocampa*. There are districts in the Kahlberger Schweiz which have been rendered almost uninhabitable by the irritative skin eruptions caused by the presence of these insects, the action of which is not necessarily in consequence of actual contact with them.

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